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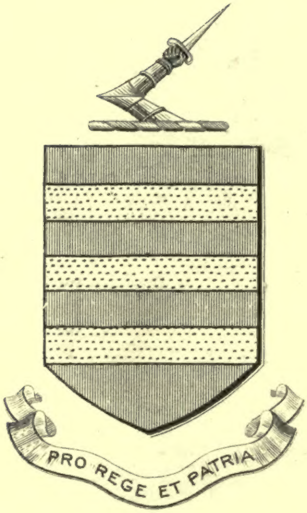
H. A. Wright



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OF THE

PRACTICE OF MEDICINE.

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EDITED BY DR. H. VON ZIEMSSEN,
PROFESSOR OF CLINICAL MEDICINE IN MUNICH, BAVARIA.

VOL. VII.

DISEASES OF THE CHYLOPOETIC SYSTEM,

TOGETHER WITH THE CHAPTERS ON

DISEASES OF THE NASO-PHARYNGEAL CAVITY AND PHARYNX,
LARYNGITIS PHLEGMONOSA, PERICHONDRITIS LARYNGEA, ULCERATIONS AND TUMORS, AND
NEUROSES OF THE LARYNX.

BY PROF. HERMANN WENDT, of Leipzig; PROF. W. LEUBE, of Jena; DR. O.
LEICHTENSTERN, of Tübingen; PROF. ARNOLD HELLER, of Kiel; PROF.
H. VON ZIEMSSEN, of Munich; and DR. A. STEFFEN, of Stettin.

Translated by

ARTHUR V. MACAN, M.D., of Dublin; EDWARD W. SCHAUFFLER, M.D., of Kansas City;
A. BRAYTON BALL, M.D., and LEWIS A. STIMSON, M.D., of New York; and J.
SOLIS COHEN, M.D., and ARTHUR VAN HARLINGEN, M.D., of Philadelphia.

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BIOGRAPHICAL SKETCHES OF THE AUTHORS.

Dr. Hermann Wendt, who died on the twenty-first of October, 1875, was the son of a plain citizen of Leipsic. He was born on the eighth of March, 1838. At first he attended the Municipal School, and later the St. Nicholas, and in 1855, when seventeen years of age, entered the University of Jena, to study medicine. A year later he returned to Leipsic, and in 1861 had completed his examinations. Having thus been equipped with a good fund of knowledge, he began to turn his special attention towards obstetrics; and, desiring to perfect himself in the practical part of this branch, spent a year in Prague and Vienna; on his return being made assistant-physician to Professor Credé at the Trier Institute. External considerations, however, opposed the plan he had formed of devoting himself entirely to obstetrics. Accordingly, in 1863 and 1864, under the advice of Professor Winter, he again visited several universities to make a special study of otology. Returning to Leipsic, in January, 1865, he was made assistant to Professor Winter, and assumed the direction of his Polyclinic for Otology. At the solicitation of Professor Wagner he devoted special study to the pathological anatomy of the ear. Favored by the excellent appointments of the Pathological Institute, the assistance of its director, and his own industry and extraordinary perseverance, he succeeded in doing more to enrich this field than any who had previously worked in it. In the year 1866 he was installed at the University as private instructor in otology, and in 1873 he was appointed extraordinary professor. The intensity and amount of his literary productions finally led, in the winter of 1874–5, to an obstinate attack of insomnia, which undermined his strength. In the spring of 1875 the indications of a severe cerebral affection became prominent, and made it necessary to remove him to the private asylum of Lindenhof, near Coswig; and here he terminated his sufferings on the twenty-first day of October, 1875.

Wendt accomplished a great deal that was good in the domain of practical otology, as his polyclinical reports show, and also his records, which were kept with the greatest conscientiousness. (Review of Cases observed in the year 1865 at my Polyclinic for Ear Diseases. Archiv der Heilkunde, 1866, pp. 382–384. Report of the Polyclinic. Archiv für Ohrenheilkunde, 1867, Band III.)

In the field of pathological anatomy and histology his productions were very extraordinary.

In the amount of work done, Toynbee alone may be compared with him; but in depth of investigation and closeness of microscopical examination, Wendt was far the superior. His very large collection of microscopical and macroscopical preparations in the Leipsic Pathological Institute is a model of arrangement, and furnishes the best proof of his work. His experimental works are as follows:

1. On Tubular Glands of the Mucous Membrane of the Middle Ear. Archiv der Heilkunde, 1870, pp. 252–257.
2. Contributions to the Pathological Anatomy of the Ear. (Cases of Cerebral Abscess, Thrombosis of the Sinuses, and Meningitis in Aural Diseases). Ibid., 1870, pp. 562–598.
3. Contributions to the Pathological Anatomy of the Ear (Diphtheria, Croup). Ibid., 1870, pp. 257–263.
4. On the Condition of the Auditory Canal and of the Naso-pharyngeal Space in Variola. Ibid., pp. 118-167. 1872, pp. 144–416.
5. On the Condition of the Middle Ear in the Fœtus and in the New-born. Ibid., 1873, pp. 97–124.
6. Polypous Hypertrophy of the Mucous Membrane in the Middle Ear. Ibid., 1873, pp. 260–274.
7. Secondary Changes, especially in the Mucous Membrane of the Middle Ear. Ibid., 1873, pp. 274–293.
8. On an apparently Embolic Process in the Mucous Membrane of the Middle Ear. Ibid., 1873, pp. 293–300.
9. Desquamatous Inflammation of the Middle Ear. Ibid., 1873, pp. 428–447.
10. On an Entothelial Cholesteatoma, with Remarks on the Histology of the Tunica Propria. Ibid., 1873, pp. 551–562.
11. On New-formed Membranes and Cords in the Middle Ear. Ibid., 1874, pp. 97–100.
12. Politzer-Kessel's Corpuscles. Ibid., 1874, pp. 120–129.

WILHELM OLIVIER LEUBE, professor of clinical medicine at the University of Erlangen, was born September 14, 1842, at Ulm, in Würtemberg. He attended the Gymnasium of that city, and in the spring of 1861 entered the University of Tübingen to study medicine. After devoting himself to the study of the natural sciences under the instruction of Vierordt, Luschka, Strecker, Hoppe, Mohl, and others, in 1863 he visited Zurich, to get an introduction into practical medicine, under Billroth, Rindfleisch, and Griesinger, of whom the latter then stood at the height of his clinical activity. Returning to Tübingen in 1864, he passed his examination under Niemeyer in 1865, obtained his degree in 1866, and then visited the Universities of Munich and Berlin, remaining in the latter city until 1868. In Munich he devoted himself especially to analytical chemistry, in Liebig's Laboratory; in Berlin to physiology under Dubois-Reymond and Rosenthal, and later to physiological chemistry under Kühne. In the spring of 1868 he took the position of first assistant at Ziemssen's Clinic in Erlangen, and was installed there in the summer of 1868 as private instructor. In 1872 he was appointed extraordinary professor, and in the same year was called to be professor of the Medical Clinic in Jena, and in 1874 in the same capacity to Erlangen.

His literary works are:

1. Experiments on the Use of the Spectroscope for the Discovery of Blood-spots. Moleschott's Untersuchungen, 1864.

2. On Localized Faradisation of the Laryngeal Muscles. Inaugural Dissertation. Tübingen, 1866.

3. Experiments on Strychnia Poisoning and its Treatment by Artificial Respiration. Dubois-Reichert's Archiv, 1867.

4. Contributions to our Knowledge of Intestinal Juices and their Action. Habilitationsschrift. Erlangen, 1868.

5. On the Pathology and Therapy of Diabetes. Deutsches Archiv für klinische Medicin, V., 1869.

6. Neuro-pathological Communications. Ibid., VI., 1869.

7. On Albumen in the Sweat. Virchow's Archiv, 1869.

8. On the Antagonism between the Secretions of Urine and Sweat. Deutsches Archiv für klinische Medicin. VII., 1870.

9. On Multiple Insular Sclerosis of the Brain and Spinal Cord. Ibid., VIII., 1870.

10. Contributions to the Question of the Cheyne-Stokes Respiratory Phenomenon. Berliner klinische Wochenschrift. 15, 1870.

11. On the Reduction in Temperature of Fever Patients by Ice-bags. Deutsches Archiv für klinische Medicin. VIII., 1871.

12. Communications on the Epidemic of Typhus in Ulm, 1870–71. Ibid., VIII., 1871.

13. On the Nourishment of Patients Fed per Rectum. Ibid., X., 1871.

14. On the Therapy of Diseases of the Stomach. Volkmann's Sammlung klin. Vorträge, 62, 1873.

15. On a New Sort of Meat Solution as a Nutritive and Curative Resort in Diseases of the Stomach. Berliner klinische Wochenschrift, No. 17, 1873.

16. Three Cases of Mycosis Intestinalis and their Connection with Splenic Fever (with W. Müller). Deutsches Archiv für klinische Medicin. XII., 1875–4.

17. On the Diagnosis of Dilatation of the Stomach. Ibid., XV., 1875.

18. Clinical Reports from the Medical Division of the Provincial Hospital of Jena. Erlangen, 1875.

19. On the Digestive Power of the Stomach Juices in Dyspepsia. Allgemeine Wiener medicin. Zeitung, 41, 1875.

20. On the Use of Compressed Air for the Filtration of Fluids. Dingler's Journal, 1876.

21. On Disturbances of Motor-Sensibility in Disease. Centralblatt für medicin. Wissenschaft, chapter 38, 1876.

Otto Leichtenstern was born on the 14th day of October, 1845, in Ingolstadt, Bavaria. After completing his studies at the High School of Neuburg, on the Danube, he entered the University of Munich, in the year 1863, and after pursuing the study of medicine and the natural sciences for six years, in 1869 passed his examination and obtained his degree as doctor of medicine, surgery, and obstetrics. From the year 1869 to 1871 he held the position of assistant physician at the Medical Clinic of Munich, under Professor von Pfeufer, and after the death of the latter, under Professor von Lindwurm. Having passed his state examination in the year 1871, he moved to Tübingen, where, during the summer semester of 1871, he was entrusted with the temporary charge of the Medical Clinic; and since that time has occupied the position of private instructor in medicine, and is first assistant at the Medical Clinic of Prof Liebermeister.

Among his contributions, exclusive of the treatise in the present volume and minor articles in journals, are the following:

On Abdominal Typhus. Inaug. Dissertation, 1871.

On Intestinal Invagination. Prager Vierteljahrschrift, Band 118, 119, 120, 121.

Experiments on the Volume of Air Expired under various Circumstances. Zeitschr. f. Biologie, VII. Band.

Notes on Physical Diagnosis in Reference to Luschka's Position of the Abdominal Organs in Man. Deutsche Klinik, 1873, No. 26-36.

The Diagnosis of Diaphragmatic Hernia. Berl. klin. Woch., 1874.

On Asthenic Pneumonias. Volkmann's Sammlung klinischer Vorträge, No. 82.

ERRATA.

On page 703, seventh line from the top, the formula should read: Picronitrate of potash, twenty-eight grains; jalap, one drachm; extract of liquorice, enough to make thirty pills; five pills three times a day.

On page 671, at the top, the title "LEECH TRIBE" should read "SUCTORIAL WORMS"; and near the middle of the same page the words "*larger type*" should read "*italics.*"

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(Translated by Edward W. Schauffler, M.D.)

DISEASES

OF THE

NASO-PHARYNGEAL CAVITY

AND

PHARYNX.

WENDT.

INTRODUCTION.

THE parts, the diseases of which are to be considered here, are included by many under the common name of pharynx. [The Germans use the two terms *Schlundkopf* and *Rachen*, both of which have to be translated pharynx, the former term literally signifying head (or top) of the gullet.—TR.] They form the walls of a cavity which is situated behind the nose, the mouth, and the larynx. This cavity, interposed between the structures named on the one side, and the cervical vertebræ on the other, extends from the base of the skull down to the cricoid cartilage, and borders laterally upon the most important blood-vessels and nerves of the neck (internal carotid artery, internal jugular vein, and the vagus, accessory, glosso-pharyngeal and hypoglossal nerves). Anteriorly it is in communication with the principal cavities of the nose, with the cavity of the mouth, and with the larynx; laterally it is in communication with the middle ear; and inferiorly it passes into the œsophagus.

Individual regions of the pharynx may be still further discriminated with advantage. Firstly, we have the soft palate and the tonsils, which have already been described in a previous division of this work. Amongst other functions, the soft palate causes a frequent separation (in the acts of swallowing and speaking) between the upper and lower portions of the pharynx. These two divisions, sometimes thus separated, while possessed of some common characteristics, present such essential differences as to their anatomical relations and physiological significance, as well as in regard to their practical bearings (their methods of becoming diseased, their diagnosis and treatment), that it seems best to consider them separately as well as to give them distinct names.

The term *naso-pharyngeal cavity*, or *naso-pharyngeal space* (*cavum pharyngo-nasale*, or *retronasale*) is almost universally employed to designate the upper portion by those who have in any way busied themselves with this region. The lower portion I shall here merely term *pharynx* or *pharyngeal cavity*, because I deem it advantageous to limit this much-employed indefinite name to a circumscribed region to which it is alone physiologically applicable.

In what follows I shall employ the term pharynx (Schlundkopf) for the entire cavity, and naso-pharyngeal space and lower pharynx (Nasenrachenraum und Rachen) for its two divisions, so different from each other.[1]

The favorite division, by many anatomists, of the lower pharynx into an oral portion (*pars oralis*) and a laryngeal portion (*pars laryngea*) has no practical value.

The diseases occurring in the pharynx are either part of the manifestations of general disease, or purely local. They are either limited to some individual portion (orifice of the Eustachian tube, pharyngeal tonsil, posterior pharyngeal wall, etc.), or they affect an entire section, or the entire cavity.

They may occur alone or in connection with simultaneous diseases of the adjacent organs. These latter may be dependent upon affections of the pharyngeal structures, or may result from them. The naso-pharyngeal space, for instance, stands in such a pathological connection with the nose and the middle ear especially; and the lower pharynx with the larynx and mouth.

The significance which these diseases may acquire principally lies in their impeding important functions (speech, respiration, swallowing, hearing).

The lower pharynx is very frequently found diseased, and is readily accessible to anatomical examination, and in part, also, to direct exploration in the living subject. Despite this, its pathology has been but little cultivated, much less than that of the palate. In a great proportion of the acute and chronic affections of the palate (the anginas) there is a participation on the part of the lower pharynx, either in its entire tract, or in individual portions, especially those that are contiguous. Its behavior with reference to anatomical alterations, method of origination, and the course of these affections accords with that of the palate, or it is modified ; the intensity of the process being often diminished, and its duration shorter or longer. This simultaneous participation of immediately contiguous parts in the same diseases, from the same causes, and for the most part with the same or similar symptoms and course, induces me particularly to refer to the thorough consideration which has already been given to the subject of anginas, rather than to have recourse to too much repetition.

---

[1] The translator will render the latter term (Rachen) by *lower pharynx*, to distinguish it from the entire *pharynx*, the resources of the English language being unequal to greater accuracy in following the author's precise meaning.—TRANSLATOR.

Another class of diseases of the lower pharynx exhibits greater independence, and it is this class to which special consideration will here be given.

Diseases of the naso-pharyngeal space are not less frequent. A knowledge of them has been especially acquired in recent times, especially by surgeons (morbid growths) and aurists (relations to the middle ear). These cavities have become more accessible by new methods of examination, and more light has been shed upon them by anatomical investigations. The period of time which has since elapsed is short, and the difficulties of investigations upon the living subject and upon the corpse are not slight. Consequently, the developments of pathology in this direction have not yet progressed very far.

Remarks on Anatomy and Physiology.

Concerning the *anatomy*, consult the various manuals, as well as *von Luschka*, Der Schlundkopf d. Menschen. Tübingen. 1868.—*Lacauchie*, Traité d'hydrotomie. Paris. 1853.Tab. II. fig. 10.—*Kölliker*, Handbuch d. Gewebel. 5 Aufl. p. 392.—*F. J. C. Mayer*, Neue Unters. a. d. Gebiete d. Anat. u. Phys. Bonn. 1842.—*C. Th. Tourtual*, Unters. üb. d. Bau. d. mensch. Schlund.- u. Kehlk. Leipzig. 1846.—*Th. Landzert*, Ueb. d. Canalis craniopharyngeus am Schäd. d. Neugeb. Petersb. med. Ztschr. XIV. 1868. 3 and 4.—*von Tröltsch*, Beitr. z. anat. u. phys. Würdigung d. Gaumenmuskeln. Arch. f. Ohrenh. Bd. 1. p. 15.—*Rüdinger*, Ein Beitr. z. Anat. u. Hist. d. Tub. Eust. Bayer. ärztl.-Intell- Bl. 1865. No. 37.—Illustrations in *v. Luschka*, loc. cit. (12 plates), in *W. Braune*, topogr. atl. (plates 3–5), in *Rüdinger*, Atlas d. menschl. Gehörorg. (Lief. II. Taf. 7 u. 8). —Methods of making sections, in *v. Luschka*, loc. cit. Text z. 5. Taf. in *Wendt*, Ueb. d. Verh. d. Gehörorg. u. d. Nasenrachenr. bei Variola. Arch. d. Heilk XIII. p. 120.

In reference to *physiology*, consult, besides the manuals, *G. Passavant*, Ueb. d. Verschliess. d. Schunds b. Sprechen. Frankf. a. M. 1863; also in Virch. Arch. Bd. 46. p. 1.—*L. Merkel*, Die Funct. d. menschl. Schlund. u. Kehlk. Leipzig, 1862; Anat. u. Physiol. d. menschl. Stimm. u. Sprachorg. Leipzig, 1863.—*J. F. Kräuter*, D. physiol. System d. Sprachlaute. Arch. f. An., Phys. u. w. Med. 1873. p. 449.—*Kussmaul*, Ueb. d. Schnupfen d. Säugl. Henle u. Pfeuf. Zeitschr. f. r. M. 3. Reihe, XXIII. 3, p. 225.—*Politzer*, Wien. Med. Woch. 1863. No. 6.—*Lucae*, Arch. f. Ohrenh. IV. p. 188.—*Ecker*, Virch. Arch. XLIX. 2, p. 290.—*Ogston*, Arch. f. Ohrenh. VI. p. 268.—*Zaufal*, ibid. p. 297.

General Anatomy.

The pharynx is only to a limited degree provided with a bony framework. Above, at its roof, it is formed of the body of the sphenoid bone, and the basilar process of the occipital bone. These bones are covered on their inferior surface with a stout layer of tense connective tissue, arranged in parallel strands, which also equalizes the differences of level of the neighboring portions of the base of the skull—the *fibrocartilago basilaris*, so-called, not because of its containing cartilage cells, which are not present here, but because of its dense construction, cartilage-like in hardness, and its almost hyaline appearance.

At the anterior portion of the upper section of the pharynx, there is, in the centre, the vomer, with its processes diverging upwards; to the side, the inner plate of the pterygoid process of the sphenoid bone; below, the horizontal plate of the palate bone; all of which together compose the boundaries of the choanæ.[1] The posterior wall, in its entire length, rests upon the cervical vertebræ, as far down as the sixth (inclusive).

The true walls of the pharynx are principally formed of three layers: mucous membrane, muscles, and the fascia surrounding them.

The muscles, which form the limit of the pharynx externally, surround it posteriorly and laterally, partly in transverse or oblique directions, and partly in longitudinal arrangement of their fibres. There are three symmetrical pairs of constrictor muscles—superior, middle, and inferior—which assist in forming the lateral and posterior walls of the pharynx, and converge in the middle line of the posterior wall. Then there are the pair of stylo-pharyngeal muscles, which take origin from the styloid process, and run down on each side, to be inserted in the wall of the pharynx, the palatine tonsil, and the larynx. (Concerning the muscles of the palate, see elsewhere, in the proper connection.)

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[1] The posterior portion of the nose, though practically belonging to the naso-pharyngeal space, must be excluded here from a thorough description.

The muscles are covered on their external surface by the *external fascia*, which in the lower two-thirds of the pharynx is formed by the deep layer of the fascia colli; while its upper third belongs to the bucco-pharyngeal fascia. Inwards, towards the mucous membrane, the muscles are covered by the internal fascia, fascia interna or cephalo-pharyngea. This runs downwards from the base of the skull as a continuation of the fibro-cartilage.

The *mucous membrane*, which covers the interior surface of the pharynx, though exhibiting some general uniformity of structure, shows important differences in various localities. In general, it is richer in blood-vessels and glands in the naso-pharyngeal space (redder, moister), while it is thicker (diffuse cytogenetic layer) and less movable here than in the lower pharynx.

The epithelium in the naso-pharyngeal space is formed of ciliated cylindrical cells. In the lower pharynx there is a stratified, flat epithelium of similar structure to that of the mucous membrane of the mouth. The division line is sometimes sharply defined, and sometimes indicated by transition forms. The cylindrical epithelium sometimes extends farther downwards (as far as beyond the region of the atlas) on the lateral and posterior walls, or the flattened epithelium extends much higher up on the posterior wall. In a number of cases I have even seen the posterior portion of the pharyngeal tonsil covered with several layers of flattened epithelium.

The stroma of the mucous membrane, also, is different in the upper and lower sections of the pharynx. In the greater portion of the pharyngeal cavity (at the roof—here more strongly developed as the pharyngeal tonsil—at the pharyngeal projections of the Eustachian tube, and in the fossa of Rosenmueller) it exhibits the composition of adenoid (His), cytogenetic (Kölliker) substance, that is to say, of the well-known network similar to that of lymphatic-gland tissue, with extensive distribution of lymphatic, nucleated cells in the inter-spaces. In the remaining portions of the naso-pharyngeal space (anterior region of the Eustachian tube, lower end of the posterior wall) and in the lower pharynx, it is composed of irregularly interwoven fibres, which in some places exhibit a denser structure in their deeper layers. There is no deposition of cells here normally, in the spaces between the fibres, and nothing like a reticulated arrangement of the latter. Apart from their occurrence pathologically (see further on), there is no elevation of the surface into papillæ in the naso-pharyngeal space, nor in the lower pharynx ; or, if such does exist, it is merely rudimentary. Kölliker has called attention to the richness of the pharyngeal mucous membrane in elastic fibres.

Follicles, roundish, circumscribed masses of cytogenetic tissue, most dense around their margin, are always found in great numbers in the cytogenetic layer of the naso-pharyngeal space (in the tonsils, at the pharyngeal projection of the Eustachian tube, in the fossa of Rosenmueller), besides being found isolated (solitary) or united, several in one group (conglobated), in the mucous membrane of the lower pharynx. Here their number varies in different individuals, being often very slight, and their distribution upon the various sections of the surface also varies much. The greatest number of follicles is usually found in the upper third of the posterior pharyngeal wall. They also occur with regularity, conglobated, in the laryngo-pharyngeal sinuses (*recessus laryngo-pharyngei*).

Tubular glands, inversions of the mucous membrane lined with epithelium, with diffuse or follicularly arranged cytogenetic surroundings, are, in the normal lower pharynx, encountered most constantly in the laryngo-pharyngeal sinuses. In the naso-pharyngeal space, the depressions of the surface, which are here quite numerous, and especially the fissures, *i. e.*, the lacunæ of the tonsils, may be included in the same category.

The mucous membrane of the lower pharynx contains numerous *acinous glands*. In the naso-pharyngeal space their number is unusually great. They here form a continuous sheet, a regular layer, about the circumference of the mouth of the Eustachian tube, in the tissue over its pharyngeal prominence, in the fossa of Rosenmueller, and especially, also, in the tonsils.

Blood-vessels.—The principal artery coming into consideration is the ascending pharyngeal, which supplies the greater portion of the pharynx and the Eustachian tube. In the naso-pharyngeal space there are, in addition, small terminal branches of the internal maxillary artery, the Vidian and the superior pharyngeal, in frequent communication with the ascending pharyngeal, in the anterior portion of the vault of the pharynx and its lateral walls.

The capillary network is tolerably dense. A return in loops is frequent near the free surface.

The veins gather themselves together in the deeper layer into a coarse wide-meshed network, which conveys its blood chiefly into the internal jugular vein.

The *lymphatic vessels* form a thick network in the mucous membrane, and a coarse plexus in the muscular tissue. The upper vessels are in communication with two lymphatic glands lying upon the posterior wall near the base of the skull, and the lower vessels with several glands in the vicinity of the greater horn of the hyoid bone (von Luschka).

Nerves.—The second branch of the trigeminus gives off pharyngeal rami to the roof of the pharynx and the parts surrounding the pharyngeal orifices of the Eustachian tubes; the third branch, to the abductor of this orifice (tensor palati muscle). The glosso-pharyngeus supplies the stylopharyngeus muscle, the superior and middle constrictors, and the mucous membrane. The vagus furnishes, in addition to the fibres for the levator (Arnold, Wolfert), two twigs which connect with those from the accessory nerve of Willis (according to Hyrtl and Rüdinger, from the glosso-pharyngeus), and pass to the upper and middle constrictor muscles and to the mucous membrane. Its superior laryngeal nerve supplies the lower constrictor. Fibres from the sympathetic connect with the pharyngeal branches of the glossopharyngeus and vagus.

The Individual Parts.

The *naso-pharyngeal space* has somewhat the configuration of a cube with strongly rounded corners in part. It may also be compared, on account of the peculiar bend of its roof, the steep descent of its anterior and widely-open wall, to the windsail of a steamer (Hyrtl), to a prompter's box, or to the roof of some old-style coach. Its average depth is two centimetres (four-fifths of an inch), its breadth between the fossæ of Rosenmueller three and a half (one and two-fifths inches), and between the projections of the Eustachian tube two (four-fifths of an inch), and its height is always more than two centimetres.

Its *roof*, the pharyngeal vault, the *fornix*, which is concave inferiorly in a horizontal direction, includes the upper and (to a great extent) the posterior wall. No distinct demarcation between these two walls is perceptible on the interior surface; they round off into one another in a shallow arch-shaped continuation. The *pharyngeal tonsil* is found here at the roof of the pharynx. The cytogenetic, richly follicular layer which, without interruption, covers the vault and a great portion of the lateral wall (recessus, projection of tube), and which composes the stroma of the mucous membrane, is, as a rule, more considerably developed at this place, and elevated into a thick cushion which may reach the thickness of six millimetres (one-quarter of an inch) or more. Its composition and form can be positively determined only upon fully normal subjects. The origin of many different views as to its construction is evidently dependent upon the employment of pathological material.

The dividing-line between the normal pharyngeal tonsil and the contiguous surface of mere mucous membrane is distinctly recognizable anteriorly (from the upper circumference of the choanæ [posterior nares]) and posteriorly (towards the upper border of the arch of the atlas). The tonsil is elevated above the mucous membrane. Below its posterior termination there remains a stripe of mucous membrane about one centimetre (two-fifths of an inch) in height, corresponding to the position of the anterior arch of the atlas, which might be designated as the *posterior wall*, in contradistinction to the roof. It is divided, by narrow or wider fissures, *lacunæ*, which sink into its substance from one-third to one-half and more of its entire thickness, into projections, crests, and ledges of varying height and breadth, which run either in a strictly horizontal direction, or converge somewhat posteriorly, seldom anteriorly. The number and arrangement, as well as the height and breadth of the prominences vary. The lateral crests are often much less high than those in the centre; and those lying on the outside border are separated by a specially deep indentation from the upper portion of the lateral walls. The crests frequently diminish in height towards the front and the rear. Among other configurations it may acquire a flattish semi-globular appearance, which makes still more prominent its resemblance to the palatine tonsils, a resemblance chiefly founded on its histological composition.

This typical longitudinal fissuring, which is already very distinctly imprinted upon the pharyngeal tonsil of the new-born babe, though only one millimetre (one twenty-fifth of an inch) in thickness, may still be recognized in various apparently deviating forms which appear, sometimes under normal conditions (delicate or irregular formation of the ridges and spaces in the development of the organ during the first years of life), sometimes associated with or in consequence of the diseases which so often occur in this region (especially tumefaction and hypertrophy, total and partial adhesions, atrophy and obliteration of the ridges). Such modified forms (see farther on) come under observation in adults more frequently than do entirely normal forms. They present a tolerably uniform surface, or (and more frequently) a remnant of the longitudinal fissuring in interrupted slits or perforations, or even a sponge-like perforated surface.

F. J. C. Mayer describes as the pharyngeal bursa (bursa pharyngea) a pouch-like depression in the posterior portion of the tonsil. Von Luschka explains it as a remnant of the canal which is produced in the formation of the cerebral appendix [glandular lobe of the pituitary body.—*Tr.*] by a constriction of the primordial pharyngeal wall (Rathke). Several large cysts at this place (Czermak, von Troeltsch) have been described in the medical literature of this region. The pouch is not constant, as has also been remarked by von Luschka. Similar depressions likewise occur at other places in the tonsil.

Landzert discovered within the sphenoid bone of the new-born child a canal ending blindly downwards, which he regards as a remnant of the communication just mentioned between the cranial and pharyngeal cavities, and which may give occasion to prolapse of the pituitary gland in dropsy of the ventricle.

On the lateral wall posteriorly are the fossa of Rosenmueller, the cushion or projection of the Eustachian tube, and its orifice. The projection is composed essentially of a strip of cartilage which passes obliquely forwards, inwards, and downwards from the tip of the temporal bone, increasing considerably in height and breadth as it does so. It is in part adherent to the fibrous cartilage at the base of the skull, and is bent, gutter-like, outwards and forwards in its entire course, constituting the median wall. The concavity of this cartilage is clothed with a mucous membrane which leaves it, at its lower border as well as at the upper end where it is rolled over, to form the lateral wall, which lies lightly against the middle wall, when in repose. This lateral wall, otherwise movable in its entire extent, and which can be abducted from the middle wall by the tensor muscle of the palate, is, at the pharyngeal extremity of the tube, tensely attached to the pterygoid process of the sphenoid bone, and is reflected back to a certain degree like a curtain, constituting the anterior lip of the tube. The strip of cartilage, on the contrary, projects obliquely into the naso-pharyngeal space, increasing in height and breadth, and composing the *tubal prominence;* it also forms the posterior lip, and, by bending in a direction upwards and forwards at the same time, forms the upper border of the *orifice of the tube.* This orifice is funnel-shaped, being oval, with a slight upward arching of its lower border, caused by the belly of the levator muscle of the palate; it is directed forwards and inwards in correspondence with the course of the canal of the tube, measuring seven to nine millimetres (twenty-seven hundredths to thirty-five hundredths of an inch) in height, and four to six millimetres (fifteen hundredths to twenty-three hundredths of an inch) in breadth.

Between the prominent tubal projection and the posterior wall, a triangular depression is left, the fossa of Rosenmueller, *recessus pharyngis*, covered with a layer of cytogenetic tissue. Its surface is often of very irregular conformation from swelling and adhesions ; otherwise it is smooth.

Instead of an *anterior wall* there are the wide oval posterior nasal orifices, the *choanæ*, separated by the vomer, and bounded externally and inferiorly by the wing-shaped processes of the sphenoid bone, and the horizontal portion of the palate bone.

An *inferior wall* is temporarily formed by the soft palate during swallowing and speaking.

The relations presented by the *lower pharynx* are very simple. Its *posterior wall* extends along the anterior surface of the cervical vertebræ from the second to the sixth. The *anterior wall* is defective above, and corresponds to the isthmus faucium ; otherwise, it is formed in the middle by the posterior surface of the larynx, and laterally by the pharyngo-laryngeal or pyriform sinuses, fossæ naviculares. Its transverse diameter considerably exceeds the antero-posterior diameter. The latter decreases very rapidly in the lower portion, with simultaneous diminution of the transverse diameter, so that, in a condition of repose, only a slit-like lumen exists, there being a complete approximation of the anterior and posterior walls in the region of the cricoid cartilage (von Luschka, von Bruns, Braune).

Von Luschka recommends for dissection in private, that the floor of the mouth should be loosened in its circumference, and the hard palate be removed, after disarticulation of the lower jaw, so as to permit of an inspection of the naso-pharyngeal cavity. In those cases in which the cranial cavity is opened, I recommend a transverse opening to be made in the posterior portion of the crista galli with the chisel, and then a portion to be sawn out upon both sides in a slightly convex arch which shall include the extremity of the petrous portion of the temporal bone, and extend as far as the anterior border of the foramen magnum. The preparation includes the entire naso-pharyngeal space, and the posterior portion of the nasal and superior maxillary cavities. Any disfigurement of the corpse is thus avoided.

Participation in Respiration, in Deglutition, and in Speech.

The naso-pharyngeal space is an important part of the respiratory tract, for breathing normally takes place principally through it and the nose. The nursling is exclusively confined to this method of breathing during the act of nursing (Kussmaul) as he also is in sleep. In increased inspiration through the nose (snuffing), and when breathing is practised exclusively through the nose, even while the mouth is open, the soft palate falls.

The lower pharynx is subservient to respiration only in its upper half. Coincident participation of the upper and lower portions takes place in the act of deglutition. The bolus, having reached the lower pharynx, is forced into the œsophagus by the combined constricting and elevating action of the constrictor muscles, while the elevation of the base of the tongue, and the approximation of the arches of the palate in front, and the soft palate above, effect a closure in these directions. A separation of the lower pharynx from the naso-pharyngeal space takes place in the formation of almost all vocal sounds, and in every movement of deglutition, whether this takes place through some influence acting from below (ingesta) or through one acting from above (nasal douche, Eustachian catheter). The soft palate, under these circumstances, always assumes a nearly horizontal position, the uvula remaining with its tip directed downwards, so that a bend or angle is formed between the two. The closure is completed by a horizontal ridge which is formed on the posterior wall of the pharynx in the region of the anterior arch of the atlas, by the dragging forward of this portion by the action of the superior constrictor muscle (Passavant).

In forced deglutition (movements of gagging) there is a very considerable constriction of the lower pharynx, and sometimes a very great elevation of the soft palate. In vomiting, solid and fluid substances frequently get into the naso-pharyngeal space; and in rare cases I have seen them gain the cavity of the osseous middle ear, having passed up through the Eustachian tube.

A free communication between the lower pharynx and naso-pharyngeal space takes place only in uttering the nasal, and the oro-nasal sounds (Kraeuter). With these sounds only do important vibrations of air in the nose and naso-pharyngeal space occur in the production of speech. In the enunciation of all other sounds the naso-pharyngeal space is closed below in the usual manner (Passavant).

The upper portion of the lower pharynx, above the entrance into the larynx, has been designated by Merkel as the "receiving tube" (Fangrohr), the function of which is to take up the sonorous current of air escaping from the larynx before it undergoes further modifications.

Relations to the Ear.

The fluctuations in the pressure of air occurring from various causes in the naso-pharyngeal space are propagated to the column of air inclosed in the cavity of the middle ear, when these fluctuations are strong enough to overcome the resistance of the walls of the Eustachian tube, which are usually in light approximation, or when an act of swallowing takes place accidentally or purposely, by which the calibre of the tube is rendered patulous. The tensor muscle of the palate dilates the tube by abducting its lateral membranous wall from its median, cartilaginous, immovable wall. The levator muscle lifts the floor of the canal and helps to give the calibre a circular form ; only the pharyngeal orifice, which is otherwise in a fixed position, undergoes a constriction by the ridge-like elevation of the lower and movable border.

The occasional wide patulousness of the Eustachian tube is necessary in order to prevent the existence of a different atmospheric pressure in the cavity of the tympanum from that of the external air, etc. Impediment or abolition of this function of the Eustachian tube (closure from swelling, or accumulation of secretion, insufficiency of the muscles) produces primarily mechanical disturbances (rarefaction of the air, sinking in and increased tension of the tympanic membrane and the chain of aural bones, and, with this impairment of hearing, subjective manifestations of sound), and consecutively anatomical alterations (hyperæmia, dropsy and swelling *ex vacuo*) in the cavity of the tympanum.

Considerable variations of atmospheric pressure take place here, especially through involuntary or intentional exaggerated respiratory movements, especially in gaping, coughing, sneezing, and nose-blowing, in the active performance of the experiment of Valsalva (strong expiration with closure of nose and mouth), and in the passive performance of the same act as suggested by the author, as useful under certain conditions, especially in the case of children (forced blowing through a tube which is inserted air-tight into one nasal orifice, while the other is kept closed). A sudden condensation of the air in the naso-pharyngeal cavity takes place, furthermore, in the escape of gases from the stomach. The air then not infrequently passes into the middle ear (with pain in inflammatory conditions of the tympanic membrane). In Politzer's procedure the act of swallowing is utilized with advantage to drive air into the middle ear, it being forced into the naso-pharyngeal space from an elastic bag, the nozzle of which is inserted into the nostril.

Finally, variations of atmospheric pressure in the naso-pharyngeal space are produced by contractions of the soft palate, similar in effect to the to and fro movements of a pair of bellows. These variations are slight when the nose is patulous, but they may become of more importance if the nasal passages are closed, whether voluntarily compressed, or occluded with secretions, polypi, etc. As the soft palate springs upwards the fluctuation is positive, and as it relaxes downwards it is negative. Both alterations of pressure are propagated along the simultaneously opened Eustachian tubes into the cavity of the tympanum, and may produce corresponding manometric movements of the tympanic membrane, recognizable through an aural speculum.

Such fluctuations of atmospheric pressure are frequently utilized by surgeons, diagnostically and therapeutically, especially by means of Politzer's method. They may, however, also exert an injurious influence on the ear. The unintentional Valsalva experiment which people undertake when blowing the nose forcibly while the nostrils are blocked (emphysematous patients with bronchial and nasal catarrh), may produce relaxation of the tympanic membrane by frequent repetition of the distention. Further, when the nostrils are occluded, the air in the middle ear becomes rarefied by each act of swallowing; and in this way a purely mechanical deafness may be produced in an otherwise healthy ear (Lucae).

It may also be mentioned that in hanging (Ecker) the base of the tongue is pressed so high upwards by the cord, that the soft palate is forced up against the choanæ, occluding them. The sudden increase of pressure thus produced is so extensive under some circumstances, that it may extend into the cavity of the tympanum by separation of the walls of the Eustachian tube, and thus produce rupture of the membrane of the tympanum, as in a case reported by Ogston. (Zaufal explains the rupture by condensation of air in the tympanum, consequent upon compression of the orifice of the tube.)

General Symptomatology; Diagnosis; and Treatment.

Symptoms. NASO-PHARYNGEAL CAVITY: *Kussmaul*, l. c.—*von Tröltsch*, Lehrb. d. Ohrenh. V. Aufl. Leipzig. 1873. pp. 301, 303, 304.—*Rühle*, Volkmann's Samml. klin. Vorträge. Nr. 6. p. 28.—*W. Meyer*, Ueb. adenoide Veget. i. d. Nasenrachenh. Arch. f. Ohrenh. Bd. VIII. p. 136.—LOWER PHARYNX: *Bamberger*, Handb. d. spec. Path. u. Ther. VI. Bd. 1. Abth. pp. 4, 10, 20, 24, 99.—*O. Weber*, in Pitha u. Billroth, Chirurgie, Bd. III. 1. Abth. 2. Heft. pp. 355, 356, 358.—*v. Luschka*, l. c. pp. 7, 113.

Diagnosis. RHINOSCOPY: *Voltolini*, Die Rhinosk. u. Pharyngosk., Festschrift, Breslau. 1861.—The same author, Die Anwend. d. Galvanokaust., etc. II. Aufl. Wien. 1872. p. 94.—*Semeleder*, Die Rhinoskopie. Leipzig. 1862. (Contains, among other things, especially good rhinoscopic illustrations, and the literature of that period).—*Löwenberg*, Die Verwerth. d. Rhinosk., etc. Arch. f. Ohrenh. Bd. II. p. 103.—INSPECTION OF THE NASO-PHARYNGEAL SPACE FROM THE FRONT: *Michel*, Ueb. d. Verh. d. Tubenmdg. Berlin. klin. Wochenschr. 1873. No. 34; Tagebl. d. 46. V. d. N. u. A. p. 169.—*Meyer*, l. c. p. 149.—DEFECTS: *Magnus*, Der Nasenrachenraum, ibid. Bd. VI. p. 246.—*Ménière*, Gaz. méd. 1857. No. 19.—*Bidder*, Neue Beob. üb. d. Beweg. d. weich. Gaumens. Dorpat. 1858.—DIGITAL EXPLORATION OF THE NASO-PHARYNGEAL CAVITY: *Meyer*, l. c. p. 153.—INSPECTION OF THE PHARYNX: *O. Weber*, l. c. p. 335; with the mirror: *Türck*, Klinik d. Kr. d. Kehlk. Wien. 1866. p. 102.—*v. Bruns*, Die Laryngosk. u. die laryngosk. Chirurgie. Tübingen. 1865. p. 73.

Treatment. GARGLING: *v. Tröltsch*, Lehrb. p. 342.—NASAL DOUCHE: *Th. Weber*, Tagebl. d. 39. V. d. N. u. A. p. 207.—INSUFFLATION OF FLUIDS: *Siegle*, Die Behandlg. d. Hals- u. Lungenl. mit Inhalat. III. Aufl. p. 105.—INJECTION: *v. Tröltsch*, op. cit. p. 338.—*Gruber*, Lehrb. d. Ohrenh. Wien. 1870. p. 261.—*Guersant*, Gaz. d. hôp. 1865. No. 12.—CAUTERIZATION: *Störck*, in Semeleder.

op. cit. p. 66.—*v. Tröltsch*, op. cit. p. 340.—*Meyer*, l. c. p. 262.—APPLICATION OF FLUID MEDICAMENTS IN THE PHARYNX: *v. Sigmund*, Zur örtl. Behdlg. syph. Mund-, Nasen- u. Rachenaff. Wien. Med. Wochenschr. 1870. Nos. 32, 34, 36, 38. —*Merkel*, Tagebl. d. 42. V. d. N. u. A.—INHALATIONS: *Waldenburg*, Die Inh. d. zerstäubt. Flüss. Berlin. 1864.—*Lewin*, Die Inh.-Therapie. II. Aufl. Berlin. 1865.—*Siegle*, op. cit.—PASTILLES: *Devreux*, Press. méd. 1866. No. 38.

Symptoms.

Alterations of Respiration and of Speech.

In some of the diseases of the naso-pharyngeal space, obstruction, or even complete arrest of breathing through the nose occurs; this is the case, for instance, in very extensive increase in volume of the parts (especially the pharyngeal tonsil), or, in slighter grades, by occlusion of the remaining portion of the calibre by secretion; it also occurs when new formations exist which acquire very great bulk, or at least occlude the posterior nares. Breathing then takes place in great part, or exclusively, by the mouth. In consequence of long continuance of this condition of things, the expression of the face becomes altered in a characteristic manner.

Breathing through the lower pharynx may be impeded (dyspnœa) by extensive phlegmonous swelling of the mucous membrane, by retro-pharyngeal abscesses, and by tumors projecting into the cavity. Suffocative paroxysms occur in case of pressure upon the larynx, and from the penetration of pus into the larynx.

The same causes which impede respiration through the nose produce a modification of speech. Speech becomes toneless on account of diminished or abolished resonance of the upper cavity, and the nasal sounds are enunciated only with difficulty or not at all, constituting speaking *without* the nose.

Large tumors in the lower pharynx (morbid growths, abscesses, extensive swelling) may produce important alterations in speech. The voice sounds as though the patient had a large bolus or a lump (O. Weber) sticking in his throat. Speech may even be in entire abeyance in this manner.

Motor Disturbances.

Difficulty in swallowing is frequent in diseases of the lower pharynx ; less frequent in diseases of the naso-pharyngeal space. It is dependent upon various affections of the mucous membrane (swelling, hypertrophy and atrophy, ulceration) and of the muscular tissue (fatty degeneration, purulent infiltration, the presence of living trichinæ, local or central [medulla oblongata] pareses), and on the presence of tumors of all sorts, which narrow the passage for the ingesta, and limit its capacity to the deglutition of fluids, or render swallowing altogether impossible. In rare cases exostoses of the bodies of the vertebræ, phleboliths in varicose veins on the posterior wall (von Luschka), may impede the act of swallowing.

Impaired movement of the head, holding the head stiff, occurs in cases of pharyngeal abscesses, especially such as originate in disease of the vertebræ (retro-pharyngeal abscess), in severe phlegmons, and in some morbid growths.

Manifestations Dependent on Anomalies of Secretion.

Quite a number of symptoms occur in connection with increased, altered, or diminished secretion.

The mucus secreted in the cavities often acquires great viscidity, and even a leathery consistence. Crusts, scabs, desiccated mucus and pus are encountered only in the naso-pharyngeal space, as a rule ; and then only when, through nasal defects, occasion is given to a more extended and often immediate influence of the atmospheric air. The copious presence of tenacious mucus gives the patient occasion, mostly in the mornings, for frequent hawking (lower pharynx) and movements of gagging (lower pharynx and naso-pharyngeal space), which may excite nausea and even vomiting.

Intermixture of fresh or altered blood with the mucus occurs frequently—much oftener without ulceration than with it.

A foul odor of the air expired through the nose sometimes occurs from decomposed, ichorous, or fetid pus secreted in the naso-pharyngeal space (in ulcerations, etc.), or in the middle ear (so, among others, in a case of Schwartze's there was a smell and taste like that of rotten egg, long continued, on injections into a wound made in trephining the mastoid process).

Ruehle and von Troeltsch, with justice, ascribe an injurious influence upon the stomach to the great quantity of secretion which flows down into it.

The masses which are encountered in these cavities, or are discharged from them, are not always secreted at the points where they are found. The mucus or pus accumulated in the naso-pharyngeal space, or gliding down the pharyngeal walls, may come from the posterior portions of the nasal passages or from the middle ear. If there is a profuse secretion in the middle ear, and the Eustachian tube is patulous, the secretion flows down through the tube—in great part even when there is perforation of the tympanic membrane. Clumps of cheesy, coagulated pus also pass in this way from the cavity of the ear, on powerful syringing of the outer ear. These may form complete cholesteatomatous masses in consequence of desquamative inflammation of the mucous membrane (cholesteatoma of the petrous bone, of authors); and in cases observed by the author, such masses were removed through the Eustachian tube by gargling. Finally, in epistaxis, in hemorrhage in the tympanum, and in fracture at the base of the skull, blood may flow into the naso-pharyngeal space in varying quantity.

Subjective Sensations.

A sensation of dryness, associated also with a sense of tension, occurs at the commencement of acute catarrhs and inflammations, but especially in atrophy and in anæmia of the mucous membrane of the naso-pharyngeal space and of the lower pharynx; in the former locality, also, when the direct access of air is facilitated by extensive nasal defects, and in the latter, when respiration is carried on through the mouth alone, in consequence of impermeability of the naso-pharyngeal space, or of the nose.

The sensation of the presence of a foreign body exists not only during its sojourn in the parts, but sometimes also continues some little time after removal of the offending substance or its passage downwards, in consequence of slight wounding of the mucous membrane.

In the lower pharynx such irritations are left by fish-bones more especially; and in the naso-pharyngeal cavities, by the use of the Eustachian catheter and other instruments.

Pains, for the most part not distinctly localized, occur in phlegmonous inflammations, but are infrequent in the severer grades of catarrhs, and exist in some cases of ulceration. They are increased when the affected parts are set in motion by the act of swallowing (pharyngeal orifice of the Eustachian tube, wall of the lower pharynx). Many persons are not posted concerning the glosso-pharyngeal nerve which supplies the lower pharynx and the (ramus Jacobsonii) tympanic cavity, so that pains dependent upon a pharyngeal affection may be referred to the sound ear, and contact with the aural sound, and cauterization of the exposed tympanic cavity through a defect in the membrane of the tympanum may be first felt in the lower pharynx.

Headache is often complained of in acute catarrhal and inflammatory processes. If the naso-pharyngeal space is the affected part, the pain is usually referred to the back part of the head. Occipital pains are also frequent in many cases of tumors in this cavity, after operations, and severe medicinal treatment, and also, again, in many cases of chronic catarrh. Von Troeltsch describes intense and also unilateral pains in chronic retro-nasal catarrh.

Symptoms referable to Adjacent Organs.

In diseases of the naso-pharyngeal space, pains in the ear occurring in deglutition, indicate participation in the disease on the part of the pharyngeal orifice of the Eustachian tube, or even of the tube itself. Hardness of hearing, subjective sensations of hearing, a sense of pressure and fulness in the ear and head, are chiefly caused by closure of the tube (rarefaction of the air); but are also often caused by extension of disease to the tympanic cavity. Hoarseness, dyspnœa, and apnœa occur on the extension to the larynx of many pharyngeal affections. Participating disease of the nose may occasion manifestations precisely similar to those produced by disease of the naso-pharyngeal space:—

headache, especially in the forehead, speech without use of the nose, breathing through the mouth, and disturbances due to the secretions flowing down from the posterior nares.

General Symptoms.

Disturbance of the general health occurs in many catarrhal cases, in phlegmonous inflammations, in croup and diphtheritis, and after severe operations.

Diagnosis.

Inspection of the Naso-pharyngeal Cavity from in Front.

It is not infrequently practicable to obtain a view of the naso-pharyngeal space from the front; the favorable cases being those in which there is sufficient breadth of one or both nasal cavities, slight development of the turbinated bones or their absence, atrophy of their superjacent tissues, and nasal defects of considerable size. Such a view is only possible without artificial aid in cases of considerable destruction of tissue. It is usually necessary to dilate the nostril with a suitable instrument (a bivalve ear-speculum, a dilator regulated by a screw), and then to illuminate the cavity by means of a concave reflector.

In favorable cases we then see either a vertical strip of the posterior wall; or, in addition, the mouth of the Eustachian tube with its projecting cushion; or the latter only (in oblique directions of the nasal canal). In cases of defects of the parts, proportionately more is to be seen. It sometimes happens that the posterior surface of the soft palate may be seen during movements of deglutition, rising up as a projecting transverse convex fold or cushion. More frequently the movements of the posterior wall can be observed during deglutition. Whatever is seen generally appears intensely red: strongly glistening in certain places (prominence of follicles), or diffusely red. The part seen is often too small in extent to be of service in forming a judgment from direct observation. But this glimpse of the posterior wall suffices to lead to negative conclusions (for example, as to the absence of an hypertrophy of the pharyngeal tonsil). Meyer and Michel describe pathological conditions discovered in this way; and a case of very extensive defective formation, which rendered the entire cavity accessible to inspection, is reported by Magnus, and similar reports have been made by Ménière and Bidder.

Portions of the naso-pharyngeal space can also be seen in cases of cleft or defect of the hard palate.

Inspection of the Naso-pharyngeal Cavity from Below.

Rhinoscopy, also termed pharyngoscopy, permits an inspection of almost all portions of the naso-pharyngeal cavity. A mirror introduced into the lower pharynx enables us to illuminate the walls of the cavity and to inspect them. Concerning the minutiæ of this method of examination, the following points only will be referred to. Its success depends principally upon the careful prevention of reflex movements by avoiding all unnecessary contact with the parts (depressing the anterior two-thirds of the tongue only; the use of a *small* mirror with a curved stem; the pronunciation of *a*, as in father, when the mirror is passed through the isthmus), and by securing the most favorable position of the uvula (forcible inspiration through the nose, enunciation of nasal sounds, and eventually waiting for the temporary or continuous fatigue of the muscles, the mirror being retained in the proper position). Some people (with wide pharynges, the victims of syphilophobia) are apparently especially created for rhinoscopy. In many patients the obstacles to its employment can be overcome. In isolated cases the examination cannot be made by the most experienced manipulator. The causes of failure are: great irritability, unfavorable anatomical conformation, and certain pathological conditions of the parts.

The pharyngeal mirror affords information especially concerning the amount of blood in the parts, the condition of the secretions, the amount of swelling, and the presence of morbid growths and of ulcers. Great circumspection is requisite in reference to deciding the grade of existing increase of volume in the parts, and especially in the diagnosis of ulcers. Swollen structures (the projecting lip of the Eustachian tube, the tonsils) always appear massive in the mirror. A tenacious yellow deposit of mucus is readily mistaken for an ulcer, especially when it presents the same appearance in repeated observations, despite the use of the nasal douche and the like. On the other hand, ulcers are often concealed beneath tenacious secretions, so that they may even remain unrecognized in the dead body, if careful cleansing of the parts is neglected.

Inspection of the Lower Pharynx.

Only one spot on the posterior wall of the lower pharynx, corresponding to the second and third cervical vertebræ, can be seen by direct inspection. Many patients (even children) know how to open the mouth wide enough and keep the tongue within it flat enough, to render the parts mentioned readily visible. In most cases it is necessary to press the tongue down with the forefinger, or with a spatula or spoon-handle (but only its anterior two-thirds, to avoid arousing reflex movements). The employment of dilating instruments is limited to operations. In saying *ah* the soft palate is raised ; in saying *eh* it is raised still farther, but the tongue is elevated at the same time. Diffuse daylight is sufficient, as a rule, for illumination. Artificial illumination is to be employed with care (on account of the sharp projection of the follicles from shadows, etc.). The deeper portions of the posterior pharyngeal wall and the pyriform sinuses may be brought into view with the laryngoscopic mirror, introduced in the usual manner with the tongue protruded and fixed.

Exploration with the Sound and the Finger.

The *naso-pharyngeal cavity* can be reached in almost all its parts by means of stout, straight, or catheter-shaped sounds, introduced through the nose, or by sounds with a longer and greater degree of curvature, introduced through the mouth and passed up behind the soft palate. Only gross alterations (larger morbid growths, etc.) may be discovered in this way, and then only in an incomplete manner.

More success attends the exploration of the parts with the finger passed into the pharynx. The forefinger of one hand (the other hand grasping the patient's head from behind) is carried to the posterior wall of the pharynx, passed between this wall and the soft palate, pressed forcibly through with its palmar surface forwards, and carried farther upwards at the same time. We are enabled, without changing the hand, to reach the posterior surface of the soft palate, the vomer and the posterior nares with the turbinated bones, the roof and the lateral walls with the orifice of the Eustachian tube, and the recessus pharyngis (fossa of Rosenmueller). Bleeding is readily excited by this examination, though the hemorrhage is slight in a normal condition of the parts. Sometimes nausea is produced, and even vomiting; and in some cases headache remains after the examination. No matter how rapidly and expertly it is performed, it is still very unpleasant for the patient, especially for delicate and feeble individuals and for children.

In this way we can only learn of alterations of conformation and volume—swelling and hypertrophy, morbid growths of not too small size and of distinct form. Deception, as to the relations of size, is much more likely to occur in this examination than in an examination with the mirror.

Digital exploration of the naso-pharyngeal cavities is recommendable in general only for those cases in which distinct symptoms (as breathing without the nose when that organ is patulous) point to an important malady localized in these parts, and in which rhinoscopy is not practicable.

The posterior wall of the *lower pharynx* down to the posterior surface of the larynx, with its lateral walls and the pyriform sinuses, can be felt with the finger and the sound. In this way swelling, fluctuation, the presence of morbid growths and of foreign bodies, may be detected. The result of the examination of the easily accessible lower pharynx and the palate should not lead us without further exploration to take for granted a similar condition of the naso-pharyngeal space. The latter may exhibit an entirely different condition (among other things, atrophy of the pharyngeal tonsil with hypertrophic development of the follicles on the posterior wall).

Treatment.

Avoidance of Injurious Influences.

Some diseases of the naso-pharyngeal cavities and the lower pharynx heal spontaneously, or take a favorable course, when it is possible to set aside the causal influences—the injurious conditions which have caused and maintained them. In reference to the lower pharynx the points principally worthy of consideration are overstraining in loud talking, smoking and drinking, the habitual use of very hot or irritating food and drink, and the breathing of an atmosphere charged with mechanical or chemical irritants. The last-named substances also pass the naso-pharyngeal space. The causes of the frequent venous hyperæmias here found, viz., diseases of the heart and lungs, are to be limited in their action by suitable management.

External Applications.

The application of leeches, the employment of blisters, inunctions with ointments, painting with iodine, cataplasms, moist warmth, and ice-compresses, are of some little value in affections of the lower pharynx only. In affections of the naso-pharyngeal cavities they are useless.

Removal of Secretions.

It is important to remove the masses of tenacious secretion, which, in many patients, accumulate in the naso-pharyngeal space, or coat the walls of the pharynx. Their presence is attended with annoying symptoms, and they embarrass the healing of the disease of the mucous membrane, and especially that of ulcers. There are two methods by which the secretions may be removed from these cavities.

Gargling.—This consists in repeated forcible movements of deglutition (movements of gagging), with indifferent liquids. These are if possible not swallowed, but are forced up again at the last moment of the act of deglutition. In this way considerable contraction of the pharynx is produced, and a powerful displacement of its superficial parts. In consequence of the pressure to which the mucous membrane is subjected on the part of the muscular apparatus, the mucus imbedded in its glands is pressed out like the contents of a comedo; and, in consequence of the displacement, the adherent viscid secretion upon the surface of the mucous membrane is rubbed off (posterior surface of the soft palate, walls of the lower pharynx, floor of the orifice of the Eustachian tube, and the tube itself).

Von Troeltsch very justly extols the remedial gymnastic significance of systematic practice of this kind, in the insufficiency of the muscles of the Eustachian tube (levator and tensor) in cases of hypertrophy of the mucous membrane.

By means of the nasal douche, and by injections into one nostril, the more detachable secretion can be driven from the naso-pharyngeal cavity out of the free nostril, and the adherent mucus may likewise be loosened and removed by repeated efforts of the kind. Concerning the *nasal douche*, so highly recommended by Th. Weber in the treatment of these parts, I have found the following points to hold true: A syphon-like tube about 100 cm. (one yard and four inches) in length, terminates in an olive-shaped nozzle of horn, etc., large enough to occlude the nostril and with a central aperture not more than three mm. ($\frac{1}{10}$ of an inch) in diameter. This is connected with a vessel of the capacity of half a litre; or, still better (ordinary irrigator apparatus), the tube is attached near the bottom of such a vessel. The nozzle is firmly placed in the nostril and the vessel is raised above the head, which must be strongly bent forwards (chin in contact with breast). The liquid (used bloodwarm and also cooler—86° and later 77° Fahr.) runs under slight pressure through one nostril into the naso-pharyngeal space, and out again through the other nostril. It is necessary to maintain quiet respiration and avoid movements of deglutition. A valve-like closure of the naso-pharyngeal space by the soft palate, aroused by the contact of the fluid from above (E. H. Weber), prevents its descent into the lower pharynx. By washing the cavities in this manner under moderate pressure, with a vertical direction of the Eustachian tubes, we avoid the frequently reported penetration of the fluid into the tympanic cavities and the frontal sinuses.

When the object is simply to remove the secretions, a weak solution of table-salt will suffice, which leaves less sensation of a cold in the head (swelling of the epithelium) than does ordinary water (Th. Weber). It is often desirable to supplement this operation by gargling. The douche should be used, as a rule, once or twice a day, seldom more frequently ; and should be continued for weeks or months, with occasional intermissions, when used for a long time. In special chronic cases, with profuse tenacious secretions, patients are obliged indefinitely to continue the habitual use of this cleansing process, which makes "the head feel free," and in many the ear also.

Inspiration of Fluid through the Nose.

By snuffling, by rapidly repeated inspirations through the nose, fluids may be drawn into the naso-pharyngeal space. We may "drink through the nose" (Siegle). This procedure, under certain circumstances, is a very useful substitute for the nasal douche, especially if children refuse to use the douche (which does not often happen), if one nasal passage should be impervious, or if the use of the douche (in rare cases) produces annoying results, such as pain in the frontal sinuses, irritation of the conjunctiva, or entrance of fluid into the cavity of the ear in cases of considerable hypertrophy of the pharyngeal tonsils.

Injection through the nose may be employed either directly—when it is required for the removal of adherent secretion or pseudo-membrane from the naso-pharyngeal space—or by means of a pharyngeal tube, or a straight or bent catheter perforated at its blind extremity. The walls of the cavities may be washed out on all sides in this manner. Injection with the syringe readily permits the penetration of the fluid into the tympanic cavities, which may injure a healthy ear, though it may be of service to a diseased ear (in general when there is an opening in the membrana tympani). If such penetration is desired, the nasal openings should be compressed after the introduction of the nozzle of the syringe (Gruber's procedure).

Injections into the lower pharynx are made almost only in cases of croup and diphtheritis. Guersant recommends for this purpose a tube, pharyngeal canula, one end of which is formed like the handle of a spoon, bent, and perforated with numerous fine apertures, while the other end is connected by flexible tubing with a syringe or irrigator.

Thermic Influences.

Very low and very high temperatures are not at all well borne by the mucous membrane of the naso-pharyngeal space. That of the lower pharynx, on the contrary, may be subjected to the influences of slowly-swallowed bits of ice, ice-cold water, and very warm drinks. It is desirable, where practicable, to maintain the fluid in contact with the pharyngeal walls for some time, by bending the head backwards and keeping the mouth wide open, and then to remove it again ; and to repeat this procedure, *the pharyngeal bath*, frequently.

Medicated Agencies.

Remedies are used for local treatment in the fluid or solid form, as powders or in the form of vapor. Before applying them, it is often necessary to free the affected surface of the mucous membrane from adherent secretion (nasal douche, gargles, wiping the posterior pharyngeal wall with a hair-pencil, or with a wad of cotton or charpie).

If solutions of medicines are to act in the naso-pharyngeal space, a small quantity, warmed, should be allowed to pass through by means of the *nasal douche* or the *pharyngeal tube*, always after previous cleansing with a large quantity of salt water. The number of medicaments suitable for use by the nasal douche is not large, on account of the frequent extraordinary irritability of the nasal passages through which they must pass. Only very weak solutions, too, can be employed in this manner (see farther on).

Gargles are preferred for the lower pharynx—*medicated gargles.* In ordinary gargling, which merely consists in slight movements of the soft palate, attended with a characteristic noise, very few points come in contact with the fluid, and then only for a very brief period. The *pharyngeal bath with medicated solutions* is of more utility. So, also, is a procedure suggested by von Sigmund, to force the medicament which has reached the pharynx back again *without* any accompanying movement of the soft palate. The method of Merkel, pouring a small quantity of the fluid over the base of the tongue, acts more upon the larynx and its immediate neighborhood. In this method, remedies are to be avoided, which, accidentally reaching the stomach, might act injuriously upon it.

For *inhalations*, the lower pharynx is a suitable locality, inasmuch as it is very readily reached by vapors. To be of any utility, these vapors must be used frequently and long at a time. Inhalations are, as a matter of course, not applicable to the naso-pharyngeal cavity.

Concentrated solutions and strongly irritant or caustic fluids are applied to the pharyngeal mucous membrane by means of a *brush* with a long stem. The naso-pharyngeal space may be reached anteriorly through the nose by a brush, which is to be introduced rapidly, with a screw-like motion. The nose always receives some of the contents of the brush. The naso-pharyngeal cavities can be reached from below by means of bits of sponge fastened to properly curved stems and saturated with the medicated fluid. The parts may be touched with more or less facility, according to the tolerance of the soft palate.

Remedial agents are blown into the pharynx in a *pulverized form* by means of a quill or a suitable tube, the diameter of which may be quite large. This method is also applicable to good advantage in the naso-pharyngeal space. For this purpose a large tube of hard rubber or of metal, bent somewhat like a catheter, is introduced through the mouth and pushed up behind the soft palate; or a thinner, similarly curved or a straight tube is introduced into one of the nasal passages. The powder is driven out of the tube by blowing with the mouth, either directly or through the medium of flexible tubing, or by pressure upon a rubber bulb attached to the tube. The substances used must be very finely powdered and be mixed with sugar and gum (see farther on), in order to secure longer contact with the surface of the mucous membrane. Moistening of the openings of the tube must be avoided as much as possible.

Lozenges, pastilles, bonbons, etc., to be retained a long time in the mouth, or to be slowly chewed and swallowed, come into employment occasionally only (see pharyngitis sicca).

Cauterization with nitrate of silver in substance, and the like, can be effected on the posterior wall of the lower pharynx with the ordinary caustic-holder of the pocket-case. It is more convenient to use a long silver probe or sound, with a roughened, button-like extremity, upon which a globule of lunar caustic may be melted. A suitable curve made in the instrument permits it to be used in reaching the naso-pharyngeal space from the lower pharynx. (W. Meyer has suggested an entire series of differently shaped metallic stems for this purpose.) A sound so armed, and protected by a catheter-like tube, may be introduced through the nose into the naso-pharyngeal space, and then be placed in action against different points in this space (Störck).

After the employment of caustic substances in solid form, and also after the use of various strong solutions, the excess of material should be removed by gargling or by the nasal douche, or, if necessary, by means of neutralizing fluids.

In introducing instruments into the naso-pharyngeal space from the front, the lower nasal passage of the aural surgeon—the wide triangular space between floor of nostril, septum, and lower surface of the lower turbinated bone—is to be utilized. In introducing an instrument through the mouth, the curved extremity is to be held horizontally at first, and passed through the palatal space during the elevation of the velum (the patient saying *ah*), and then its tip is to be elevated. The elevation of the palate and the depression of the tongue facilitate the manœuvre in the lower pharynx.

Operative Procedures.

The principal operative procedures are the opening of abscesses and the extraction of morbid growths. (See farther on.)

General Treatment.

In many acute diseases of the parts the fever that exists requires therapeutic attention. In many chronic cases attention to constitutional conditions is necessary. In syphilitic affections specific treatment is for the most part the main indication, even although it may never render local treatment indispensable. With many patients measures calculated to favor improved nutrition are of great importance. The onset of bettered nutrition favors the cure of many a local malady. In this respect, a residence at various watering-places in the country or a change of climate not infrequently effects a beneficial result. In many "cures" this advantage is supplemented by suitable local treatment of the affected mucous membranes.

Prophylaxis.

The disposition to recurrences and to exacerbations, which is exhibited in catarrhs of the lower pharynx and naso-pharyngeal space, renders it desirable for patients thus disposed not only to avoid all injurious influences in general, but to guard especially against the principal one—"taking cold," by systematic hardening of the skin (cold washing and rubbing, cool tub-bathing, river-bathing, or sea-bathing, and the copious use of fresh air).

The more Common Anatomical Alterations.

Smith, Consumption, its early and remediable stages. London. 1862.—*Bernard*, Lancet. I. 1852, June 25th.—*O. Weber*, loc. cit. p. 249.—*B. Wagner*, Beiträge zu d. Krank. d. Pharynx. Diss. Leipzig. 1864. (also Arch. d. Heilk. 1865).—*von Troeltsch*, Virchow's Arch. Bd. XVII. p. 78.—*Same author*, Lehrbuch d. Ohrenh. pp. 298, 299.—*Wendt*, Arch. d. Heilk. XIII. p. 433.

Anæmia.

Diminished supply of blood is a frequent attendant upon the atrophic conditions, which are to be discussed farther on. The otherwise normal mucous membrane often appears also poor in blood in general anæmia, and in persons debilitated by chronic or acute diseases, losses of blood, or suppuration. (According to Smith, anæmia of the pharyngeal mucous membrane is almost always present in the earliest stage of phthisis, and often associated with hyperæsthesia). Anæmia is usually associated with diminished secretion, and always with a corresponding loss of color in the mucous membrane. The pallor is very strongly marked under certain circumstances, so that the parts present a discoloration varying from a pale yellowish red to a pale yellowish color, and, in extreme cases, even a whitish color. These conditions disappear with improvement of the general condition; or, remaining, they lead continuously to atrophy (rarefaction).

Very extensive anæmia of the pharyngeal structures may occur rapidly, in an acute manner, in fainting and collapse; and the mucous membrane may thus acquire the color of white paper, and also evince very considerable local diminution of temperature. I have frequently been accidentally made cognizant of the commencement of fainting during an examination of the pharynx, by the coldness and pallor of the mucous membrane.

Hyperæmia.

Excess of blood in the most varying degrees is very frequently observed in the mucous membrane of the pharynx and nasopharyngeal space. It may be temporary or permanent; it may exist alone, or as is more usual, associated with increased secretion, often with swelling and other acute processes in the parenchyma; or as the source of chronic disturbances of nutrition. Many things come into consideration as causes of the notorious frequency of hyperæmia of these parts, these causes acting singly or in various combinations, in given cases. The first one to be thought of is the direct influence of irritants. Smoke, dust, and the numerous other admixtures of the air, which most men breathe, exert an influence upon the mucous membrane of the naso-pharyngeal cavities and the upper portion of the lower pharynx, which is not to be underestimated. The various irritating ingesta, in addition, come in contact with the lower pharynx. In the acute exanthemata, especially small-pox, in erysipelas, typhoid fever, pyæmia and other severe affections attended with fever, hyperæmia of the pharyngeal mucous membrane is almost always met with, often of a high grade, and usually associated with swelling, especially of its cytogenetic layer. Although this is regarded as a local manifestation of the disease, in the acute exanthemata, it is not to be forgotten that here also, as in many other affections attended with fever, the augmentation of the blood pressure by the increased activity of the heart must play an important part.

Increased tension in the aortic system also leads in another way to hyperæmia of the structures, the pharyngeal tonsil above all. In Bright's disease especially I have seen this constantly overfilled with blood to a great degree, and often strewn with hemorrhages also ; and no less so in one case in which, in consequence of the absence of one kidney, hypertrophy of the left ventricle had ensued. I have also detected pronounced hyperæmia of arterial origin in cases of phosphorus poisoning.

Obstruction in the upper *vena cava* regularly produces venous hyperæmia of the mucous membrane ; so, also, do insufficiency of the mitral valve, emphysema, and other affections of the lung impeding the circulation, especially compression of the lung by exudation into the pleura, and an elevated position of the diaphragm (long-continued meteorism in intestinal obstruction, accumulation of fæces, etc., ascites, exudation into the peritoneum, and even advanced pregnancy).

It also takes place in those who have been hanged or drowned ; also after long and repeated vomiting. Pressure of morbid growths upon the trachea produces venous hyperæmia in a marked degree. It is also always present in drunkards. In several cases thrombosis of a cerebral sinus has resulted, amongst other things, in obstructive hyperæmia and swelling of the pharyngeal tonsil.

Acute arterial hyperæmia sometimes lends the mucous membrane an appearance varying from a diffuse rosy redness to a deep red coloration. The cytogenetic structures in the naso-pharyngeal space may acquire a vermilion tint (*e. g.*, in poisoning by phosphorus). Extensive venous engorgement produces a reddish blue tint in the pharynx, and prominence usually of a coarse reticulum. The upper cavity often appears tinged an intense bluish red, even brownish red or blue.

Hyperæmic conditions are encountered in the naso-pharyngeal space much more frequently than in the lower pharynx. In the lower pharynx, too, they seldom reach so high a grade as they do in the naso-pharyngeal region in the acute exanthemata (in which diseases they are encountered as a regular thing) or in venous obstruction. Their occurrence is favored in general by the vascular richness of both sections of the pharynx, by the loose attachment of the posterior wall and the greater portion of the lateral walls of the lower pharynx, and by the lax condition of the cytogenetic layer in the naso-pharyngeal cavity. The conditions of the discharge, on the contrary, are widely different in the two sections. The discharge is facilitated in the lower pharynx by the vertical direction of its walls, and by the frequent contractions of the constrictor muscles, while it is impeded in the greater portion of the naso-pharyngeal space by the lack of any alteration in its dimensions from muscular contractions, and is especially interfered with as regards the pharyngeal tonsil, from the position of this structure—hanging down as it does from the roof.

Hemorrhage.

Hemorrhages from the naso-pharyngeal space are frequent. They are either bleedings from the surface of the mucous membrane or in the lacunæ and cavernous spaces formed by them, or they originate in the stroma itself.

The secretion, in various acute affections, accompanied with hyperæmia of a high grade (especially in small-pox, erysipelas, diphtheritis), and also in catarrh from obstruction, often exhibits a copious admixture of blood, which is sometimes altogether in preponderance—the *hemorrhagic catarrh* of erysipelas, and of many forms of variola. I have seen a copious discharge of blood from the naso-pharyngeal cavity in some cases of especially severe hyperæmia (laryngeal croup, diphtheritis of the tonsils of the palate and of the naso-pharyngeal space itself, small-pox), even without simultaneous hemorrhagic infiltration. There was always a fresh clot of considerable dimensions which adhered firmly to the parts ; and in two instances it represented a complete mould of the upper half of the cavity. A greater quantity of blood had evidently been lost than had accumulated in the clot.

Under various conditions of circulatory disturbance, small hemorrhages occur, often, in the substance of the pharyngeal tonsil, and less frequently in the coverings of the lateral walls. Extended hemorrhagic infarction also is frequently encountered here, and again at the vault especially, principally in small-pox, diphtheritis, and in collateral hyperæmia of the contiguous mucous membranes in such affections ; and also in some cases of venous obstruction. The parts severely affected undergo a discoloration varying from bluish red to brownish black, and an increase of volume, which, according to the locality, may be of (temporary) importance. Thus I have several times seen a plug-like closure of the pharyngeal orifice of the Eustachian tubes, from an infiltration of the anterior or posterior lips, like that of hæmatoma.

As regards the diseases especially predisposing to hemorrhage, I have always found diffuse hemorrhages into the parenchyma of the pharyngeal tonsil, besides intense hyperæmia, in all cases of scorbutus, and in all cases (with one exception) of Werlhof's disease and of mycosis. In phosphorus poisoning there were very numerous little exudations of blood, like petechiæ.

Isolated small hemorrhages occur in the mucous membrane of the lower pharynx under similar conditions as have been described in connection with the superior cavity ; more copious ones occur in severe forms of catarrh, and in phlegmonous inflammation. Any collection of free blood, or the formation of large clots, is here prevented by the movement of swallowing, among other things. Bernard has reported a severe arterial hemorrhage from the lower pharynx, from an extensive but not clearly described ulceration of its walls, rendering the ligation of the common carotid artery necessary.

I have sometimes found rusty-colored, brownish, or slate-colored, and even blackish, smaller or larger patches in the fossa of Rosenmueller (among other cases, one in a child four months of age), in the pharyngeal tonsil (in one instance this was of blackish gray color throughout), and on the posterior wall of the lower pharynx. A similar pigmentation has also been seen in cicatrices. The presence of great quantities of the coloring matter of the blood (hæmatoidin, melanin) showed that they were the remains of old hemorrhages. In some cases fresh discharges of blood were likewise observed.

Œdema.

Collateral œdema is not infrequently encountered about the circumference of ulcers and abscesses, as well as over them. An œdematous swelling of the mucous membrane occurs also in a very pronounced manner and to a greater extent, principally in connection with dropsical effusions in other organs, in diseases of the heart, lungs, and kidneys. Œdema of the lower pharynx, associated with difficulty of swallowing, and lasting for some days, also occurs sometimes after fractures of the upper jaw (O. Weber).

The œdema is characterized by the copious escape of fluid from the cut surface of those parts in the naso-pharyngeal space which are covered with cytogenetic tissue; and also by an unusual softness, amounting even to a glutinous consistency. That portion of the posterior wall of the pharynx which is accessible to examination appears pale, smooth, lustrous, and swollen, and retains the impression of the finger.

The serous infiltration is seldom very great in the lower pharynx. I have seen it present to a very high degree in the naso-pharyngeal cavities, in some cases, especially in two cases of obstruction (compression of the lung by exudation in the pleural cavity). In one case there was great œdema of the pharyngeal tonsil, the recessus, the tubal openings and their projections, with extensive flattening of these projections by the contiguous structures (with pointed, comb-like projections of their coverings). In the other case there was infiltration of the tonsil and the tubal projections, with occlusion of the tubal openings. It is clear that the laxity of the cytogenetic tissue and the position of the pharyngeal tonsil, suspended, as it were, from the roof, must favor venous obstruction, as well also as interstitial serous effusion.

Parenchymatous Swelling.

The mucous membrane of the pharynx frequently undergoes an increase of volume, which is chiefly dependent upon dilatation of the vessels, and more copious saturation of the tissue with blood. New cell-formation often takes place also in the cytogenetic layer of the naso-pharyngeal space, and in the follicles of the lower pharynx (catarrhal inflammation). In some cases the increase of cells in these parts is very considerably augmented ; sometimes it even amounts to a more or less thick layer of cells in the connective tissue of the mucous membrane of the lower pharynx, with a separation of its interspaces, so that a somewhat reticulated appearance ensues (*phlegmonous*, purulent, abscess-forming, *inflammation*).

These alterations (catarrhal swelling or suppuration) affect either the simple connective-tissue (lower pharynx, anterior tubal region) and cytogenetic (greater portion of the naso-pharyngeal space) basis of the mucous membrane, together with the follicles, where the latter exist, or else the participation of the follicles is in preponderance—*follicular inflammation ;* also called, on account of the prominence of the superficial follicles, *granular*, catarrhal, and suppurative *inflammation.*

The increase in thickness in the wall of the lower pharynx is only sometimes more strongly pronounced in phlegmonous inflammation. The mucous membrane is then smooth or corrugated, or disposed in folds, and only exceptionally (in abscess) strongly projected forwards. The swelling reaches a higher grade much more frequently in the naso-pharyngeal space, especially in the tonsil. The alterations in form which the parts undergo on account of the swelling of the mucous membrane are of interest. They will be referred to again in the description of retro-nasal catarrh. Here we shall only notice the great projection of the prominent structures (tubal prominence and combs of the tonsil—the latter often arranged in intestine-like convolutions), and the constriction of the cavernous depressions, even to complete occlusion (tubal outlets, Rosenmueller's fossæ, as well as the slit-like spaces between the longitudinal ridges of the tonsil, the lacunæ). In a few cases the swelling is so extensive that superficial gangrene, in the contiguous surfaces thus compressed (recessus), may ensue from pressure (in small-pox and in diphtheritis).

Hypertrophic and atrophic conditions may be developed subsequently, or a formation of callous connective tissue, like cicatricial tissue, may ensue after suppuration. In most cases there is a return to the previous normal condition, except that the original conformation—the arrangement in longitudinal ridges—is often obliterated or modified. As a result of their compression against each other, the swelling readily gives occasion to adhesions of the comb-like projections over a great extent in single or more numerous portions, with complete obliteration of the lacunæ, or leads to their subdivision into many small fissures and spaces of the most varying forms, often intercommunicating, and for the most part remaining in connection with the free surface. These cavernous spaces, which may be in part likened to follicular glands, and in part to cystoid formations, are in part true cysts (see farther on). In the fossa of Rosenmueller a reticulum frequently remains, for the most part composed of cytogenetic bridges and strands connected together. They result from adhesions of individual or more numerous parts readily ensuing upon swelling which has caused both surfaces of the recessus to lie in immediate contact.

Increased and Altered Secretion.

The secretion of the acinous glands so numerously present, especially in the naso-pharyngeal space, is increased to a very considerable degree in many affections of the mucous membrane. The secretion coats the surface in a layer often of considerable thickness. Less frequently a thinner, purulent yellowish gray mucus is produced; in most cases it is a glassy, clear or gray, yellowish white, yellow, or even greenish secretion; usually it is very viscid, and may even adhere to the parts like a firm leathery mass. Blood is often commingled with it. Not infrequently there is a dilatation of the acini, and of their excretory ducts and orifices. The mucous membrane may present a sieve-like appearance (numerous fine perforations in regular order, excretory orifices), or an appearance of small circumscribed, flattish semi-globular projections (groups of acini) distributed over the surface. The latter are similar, macroscopically, to groups of follicles, and the openings resemble the mouths of the ordinary follicular glands. Such a condition occurs chiefly on the posterior wall of the lower pharynx, and in the immediate circumference of the tubal projections.

Cysts are frequent, and cyst-like formations still more frequent; the former being closed spaces, the latter spaces still in communication with the surface, both being of varying form and size.

The cysts are due to closure of glands or portions of glands, or of lacunæ or sections of lacunæ, or they are formed from the follicular glands of the lower pharynx. It is the pressure exercised in swellings and in hypertrophic conditions which obliterates the follicular glands and the fissures of the lacunæ at some given point. The continuance of the secretion of the glands dilates the closed spaces into roundish cavities. The transformation of racemose glands into cysts is effected by the same cause, and, in addition, by the closure of their mouths and excretory ducts, from increase of the epithelium or suspension of its discharge, and by distortion of the ducts, or of glandular lobules in atrophic contraction, or in cicatrization of the contiguous parts. The true cysts, in the naso-pharyngeal space also, are for the most part provided with squamous epithelium.

The cystoid cavities are formed from normal (tubular glands, lacunæ), or from similar newly formed depressions of the mucous membrane, due to hypertrophic enlargement in ridges, or to swelling of the membrane. The temporary obstruction, by pressure, of their channels of communication with the external surface, suffices, in connection with the continued secretion and its accumulation, for a time to transform the fissures of the lacunæ of the tonsil, the narrow calibre of the tubular glands, or the pathologically formed folds of the posterior wall of the lower pharynx, into larger and even roundish hollow spaces; or to so transform them for good, if the cause has operated for a considerable time. Their production, therefore, is similar to that of many cysts, only that it does not proceed to actual growing together, to a *permanent* occlusion. They may also undergo a very considerable dilatation, in course of time, if the orifice still remaining is very minute. They often occur repeatedly in the pharyngeal tonsil, and are always clothed with the epithelium appropriate to the locality.

The contents of the cavities, as a general rule, are not influenced in their composition by the fact of their being occluded or not. We find in them (whether cysts or cystoid-spaces) either mucus of varying consistence and color, or colloid material in great coherent masses; and sometimes (also in open cavities and fissures) a fatty detritus, such as is met with in atheromata (with characteristic needles of fat, cholesterine, and chalk also, in molecular distribution or in concretions). Such masses, especially lumps of mucus and colloid matter, often escape through little holes, when pressure is made upon the pharyngeal tonsil, in unexpected quantities.

These formations are frequently met with, in both forms, in the naso-pharyngeal space. I often see them reach a size of 4 or 5 mm. (one-fifth of an inch); sometimes of one cm. (two-fifths of an inch); seldom a size of one and a half cm. (three-fifths of an inch). They are comparatively rare in the lower pharynx. B. Wagner has described several cystic formations in this region (posterior wall), of which two of the larger ones (as large as a small cherry or hazel-nut) contained detritus and cholesterine.

The Most Important Diseases.

CATARRH.—Naso-pharyngeal Cavity: *von Troeltsch*, Lehrbuch. pp. 285, 289, 299.—*Löwenberg*, loc. cit.—*Lewin*, loc. cit. p. 312.—*Berthold*, Ueber Retronasalkatarrh. Diss. Leipzig. 1870.—*The Author*, Arch. d. Heilk. XIII. p. 434.—Concerning the complications in aural diseases, see the manuals of aural diseases already mentioned.—Lower Pharynx: See the Treatises on Throat Diseases, and the references under Catarrhal Angina.—In *Bamberger*, op. cit. p. 2 et seq. will be found the references to earlier authorities, and a careful clinical description of pharyngeal affections in particular.—Pathological anatomy in *B. Wagner*, op. cit.—Fatal termination in *Bamberger*, loc. cit. p. 6; and in *Rühle*, op. cit. p. 31.

PHLEGMON.—Naso-pharyngeal Cavity: *Wreden*, Ein Fall von Verbrennung, etc. St. Petersburg med. Zeitschr. 1870. p. 6.—*The Author*, l. c. p. 435; Ueb. d. complic. Ohrenkh. pp. 130, 156.—Lower Pharynx: Consult the manuals, and under phlegmonous angina.—*Bevan*, Dubl. Journ. XLII. Nov. 1866. No. 84.—*Thiessen*, Journ. f. Kinderkrkh. XLVIII. Jan. u. Feb. 1867. No. 79.—*Stroppa*, Gaz. Lombarda. 1871. No. 35.

PHARYNGEAL ABSCESS.—*Londe*, in *Bamberger*, l. c. p. 12.—RETRO-PHARYNGEAL ABSCESS: *Hj. Abelin*, Ueber Retropharyngealabsc. b. kl. Kindern. Nord. med. Ark. III. 1871. Nr. 24.—*Desprès*, Gaz. des hôp. 1873. No. 32.—*Stephen Smith*, Am. Jour. Med. Sci. N. S. CXXIV. Oct. 1871. p. 338—*Thomas Stretch Dowse*, Med. Times and Gaz. 1871. July 8.—*Rothrock*, Phila. Med. Times. III. 1872. No. 51. Oct. 19.—*Halton*, Dubl. Journ. XLVII. 1869. May. No. 94.—*Scholz*, Wien. med. Presse. VI. 1865. Nr. 25.—*Gaupp*, Würtemb. Corr.-Bl. XL. 1870. Nr. 23.

CROUP AND DIPHTHERITIS.—Naso-pharyngeal Cavity: *E. Fournié*, Gaz. d. hôp. 1870. No. 79.—*Wreden*, Monatsschr. f. Ohrenh. 1868. Nr. 10.—*The Author*, l. c. p. 433; and Arch. d. Heilk. XI. p. 258.—Lower Pharynx: See literature under Croupous and Diphtheritic Angina.—*B. Wagner*, l. c. p. 11.

CONDITION IN TUBERCULOSIS.—Naso-pharyngeal Cavity: Verh. d. Ohrs, *v. Tröltsch*, Arch. f. Ohrenh. Bd. IV. p. 133.—*The Author*, Arch. d. Heilk. XI. p. 566.—*Schwarze*, Arch. f. Ohrenh. Bd. VI. p. 176.—Lower Pharynx: *Smith*, l. c.—*Green*, A practical treatise on pulmonary tuberculosis, New York. 1864.—*Bryk*, Wien. med. Wochenschr. XIV. 1864. Nr. 42-44.—*Isambert*, l'Union, 1872. Nos. 3-6.—*Rindfleisch*, Lehrb. d. path. Gewebelehre. Leipzig. 1869. p. 310.—*B. Wagner*, Tuberc. u. Papill. d. Pharynx. Arch. d. Heilk. VI. p. 540.—*E. Wagner*, D. Tuberkelähnl. Lymphadenom. ibid. XII. pp.1-25. 1871.—*O. Weber*, l. c. p. 360.—*Türck*, op. cit. p. 376.—*Lewin*, l. c. p. 322.

Condition in Syphilis.—See the manuals on syphilitic affections, in relation to syphilis of the lower pharynx, and also to syphilis of the soft palate.—Naso-pharyngeal Cavity: *Semeleder*, l. c. pp. 24 and 56.—*Türck*, in *Semeleder*, p. 60.—*Gruber*, l. c. pp. 572 and 574.—*Virchow*, Arch. f. pathol. Anat. Bd. XV. p. 313. *Otto*, Seltene Beob. z. Anat. Phys. u. Path. Breslau. 1816. p. 3.—*Lindenbaum*, Ein Fall v. Verwachs. d. Rachenmdg. d. Ohrtromp. Arch. f. Ohrenh. Bd. I. p. 295 (containing the literature up to that time).—*Wilde*, Practical Observations on Aural Surgery, London, 1853, translated into German by *E. v. Haselberg*, Göttingen. 1855. p. 419.—Aural Affections in Syphilis: *Schwartze*, Arch. f. Ohrenh. Bd. IV. p. 253.—*Gruber*, Wien. med. Presse. 1870. Nos. 1. 3. u. 6.—Lower Pharynx: *Trendelenburg*, Arch. f. klin. Chir. XIII. 2. p. 335.—*Ried*, Verwachsg. d. Gaumensegels mit d. hint. W. d. Rach. Jen. Zeitschr. f. Med. u. Naturwiss. I. 4. 1864. p. 409.—Treatment: *v. Sigmund*, l. c.

Hyperplastic Catarrh.—Naso-pharyngeal Space: *Czermak*, Kehlkopfspiegel. II. Aufl. 1863. p. 125.—*Semeleder*, op. cit. p. 46.—*Türck*, in *Semeleder*, p. 61.—*Voltolini*, Galvanokaust. II. Aufl. 1872. pp. 214, 219, 221, 232, 235, 237, u. Monatschr. f. Ohernh. VIII. 1874. Nr. 4.—*Löwenberg*, l. c.—*Meyer*, l. c.; Hospitals Tidende. Nov. 4. u. 11. 1868 (referred to in Schmidt's Jahrhb. CXLI. p. 325), and Med.-Chir. Transactions. Vol. LIII.—*Michel*, Tagebl. d. 46. V. D. N. u. A. p. 169.—Lower Pharynx: *Semeleder*, Die Laryngosk. u. ihre Verwerth. Wien. 1863. p. 55.—*Lewin*, l. c. pp. 364. 365.—*v. Troeltsch*, Lehrb. p. 287.—*Michel*, Zeitschr. f. Chir. 1872. II. p. 154.

Dry Catarrh.—Lower Pharynx: *Lewin*, l. c. p. 320.—*v. Tröltsch*, Lehr. p. 292.—*v. Luschka*, l. c. p. 105.

Morbid Growths.—Naso-pharyngeal Cavity: See the manuals of surgery.—Discussion in the Academy of Brussels, Presse méd. 1864. Nos. 26–29.—*Schuh*, Wien. med. Wochenschr. IV. 1865. Nos. 99–101.—*Asché*, Schmidt's Jahrhb. Bd. CLX. p. 162 et seq.—Lower Pharynx: *W. Busch*, Ann. d. Berl. Charité. Bd. VIII. 1, 1857.—*Wünsche*, Ueb. Pharyngealgeschw. Diss. Leipzig, 1864.—*v. Luschka*, Virch. Arch. Bd. I. p. 161.—*Sommerbrodt*, Ibid. Bd. LI. p. 136.—*B. Wagner*, Tuberc. u. Papill. d. Pharynx, Arch. d. Heilk. VI. p. 540.—*Mosler*, Pharyngitis u. stomatitis leukæmica. Virch. Arch. Bd. XLII. p. 444.—*Fischer*, Wien. med. Wochenschr. XV. 1865, No. 61.—*Holt*, from the transactions of the Path. So. part 5. Reference in Schmidt's Jahrhb. Bd. CXXXIX. p. 131.—*Voltolini*, Galvanokaust. p. 226.

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ARTIFICIAL EMPHYSEMA.—*Turnbull*, in *Frank's* prakt. anleit. z. Erk. u. Behdlg. d. Ohrenkrank. Erlangen, 1845. p. 173.—*Voltolini*, Monatsschr. f. Ohrenh. VII. 1873. Nos. 1 u. 10.

Acute and Chronic Catarrhal Inflammation of the Naso-pharyngeal Cavity; Acute and Chronic Retro-nasal Catarrh.

Acute Retro-nasal Catarrh.

This disease seldom occurs alone as a primary affection. More frequently there is acute catarrh of the nasal passages or of the lower pharynx at the same time, or of both these cavities, or of the larynx and the bronchi. "Catching cold" is in most cases adduced as the cause of the disease. In different stages of syphilis, usually the early stages, a subacute inflammatory condition occurs here which often progresses without any specific alterations, and which cannot then be distinguished in any manner from an ordinary catarrh. In some cases I have seen a catarrhal inflammation limited to the naso-pharyngeal space, after the insufflation of a strong powder of nitrate of silver (1:4). I have found acute catarrh of this region (on the dead subject) an almost constant lesion in typhoid fever, measles, scarlatina, small-pox, and erysipelas, as well as in croup and diphtheritis of the contiguous mucous membranes; and frequently, also, in inflammatory affections of the respiratory organs, and in several other diseases.

Anatomical Alterations.—The vascular richness of the parts, and the lax arrangement of the cytogenetic layers, readily favor an intense hyperæmia, which gives the parts a very bright color, a redness in different shades, deepening to a dark copper tint. Great swelling of the cytogenetic tissue may disguise the hyperæmia, so that the pharyngeal tonsil appears only of a dirty red color, and the tubal projections appear grayish red. In severe cases the tubal orifice, as seen in the rhinoscopic mirror (the tightly adherent anterior lip and a portion of the anterior wall), participates uniformly in this coloration. Otherwise this region appears somewhat less distinctly colored. The coloration rarely extends over the inferior third of the tube.

The same conditions which favor the engorgement with blood also favor the occurrence of hemorrhages. These take place from the surface, sometimes very copiously (see above), or they take place in the substance in small foci, or in the form of diffuse infiltration. When the free and interstitial bleeding, in connection with extensive hyperæmia, preponderates over the other manifestations, as in many cases of small-pox, in erysipelas and in pyæmia, the designation of *hemorrhagic catarrh* may be employed.

Swelling is constant, and principally in the cytogenetic layer (serous infiltration; usually also increase of cellular elements). It affects the mucous membrane uniformly in its entire extent, or it is limited to individual localities (tonsil, tubal prominence, recessus), or is especially strongly manifested in one of these.

In the milder grades of the affection there is usually only a moderate thickening of the mucous membrane. It appears more succulent, and the structures covered by it acquire a more massive appearance. The pharyngeal tonsil, swollen to the size of a centimetre ($\frac{4}{10}$ of an inch), is more sharply elevated from the upper boundary of the choanæ, and from the posterior wall. The tubal prominences project further forwards, and their orifices remain for the most part of normal size.

In severer grades of swelling the increase of volume which the mucous membrane thereby undergoes, produces very striking alterations in the configuration of the cavities which are lined by it. The pharyngeal tonsil may acquire a thickness of from one to one and a half centimetres ($\frac{4}{10}$ to $\frac{6}{10}$ of an inch), and even more in rare cases, so that it projects throughout a great portion of its extent (under certain circumstances as much as two-thirds), and considerably reduces the calibre of the space. It thereby, among other things, acquires the form of half a spheroid broadly seated upon the vault, or it surrounds the upper portion of the vomer in a heart-shaped form cut out anteriorly. In addition to this, the most different combinations in reference to the configuration of the pharyngeal tonsils are produced by the swelling. The longitudinal ridges of this organ are still distinctly observed on removal of the never-failing layer of mucus, if its typical structure has not been already destroyed by previous diseases. They either maintain their original course, only being fuller, higher and lying against one another, or else, particularly in case of very great swelling, they may be thrown into convolutions, like those of the intestines (often with an alternate narrowing and widening of the surface, according as it lies on the height or in the hollow). In cases of the latter kind the tonsil acquires a cauliflower appearance, looking as if divided up into small roundish lobes. The central portion is almost always the most thickened, and the comb-like processes are here the highest. In spite of extensive increase in the size of the tonsil in general, the choanæ often remain free (laterally) to a considerable extent, and so do also the orifices of the Eustachian tubes.

The tubal orifices frequently participate in the swelling, in severe affections. They undergo, thereby, either a slit-like or concentric constriction; or the swollen mucous membrane is raised into folds or uvula-like projections. The swelling at the anterior border is often inconsiderable (tightly adherent). A complete occlusion of the orifices themselves does not readily occur. The swelling is not sharply limited here; sometimes it extends along the inferior third of the Eustachian tube. The remaining portions of the tube participate in the swelling only exceptionally.

The cytogenetic covering of the tubal prominences often participates very prominently in the increase of volume. It then projects into the cavity much farther than usual; and in extremely swollen conditions (principally in small-pox) may undergo a lateral flattening, a triangular compression with conical points forwards and inwards, as a result of the combined pressure of the pendent pharyngeal tonsil and the swollen tissue of the recessus.

The cytogenetic clothing of the fossa of Rosenmueller is often so augmented in volume that there is close contact of its posterior and anterior walls, or even complete occlusion of the fossa.

The numerous follicles are often swollen, those which are superficial being prominent, so that tonsil, tubal prominence and recessus may appear as if bestrewn with little granules, nodules, or flat, roundish elevations, varying in size up to that of a lentil (*acute follicular*, *granular retro-nasal catarrh*). These present either the same coloration as the parts about them, or they have a gray, or grayish yellow color. Their enlargement is dependent upon an increase of their cellular elements in various degrees. If this cell-formation or deposit is very extensive, the follicles have a yellow or whitish appearance (*suppurative follicular catarrh*). Their disintegration then readily takes place, with the formation of small, roundish ulcers, and small, circumscribed, even confluent abscesses in the parenchyma, the cicatrization of which may give rise to numerous constrictions and to irregular configuration of the surface.

In many cases, on the contrary, the follicles do not undergo any important alteration; that is to say, there is a condition which does not differ from that of the inter-follicular substance.

The secretion of the mucous glands is always increased. In consequence of this, the walls of the cavities are covered with a thick layer, often sanguineous, but usually of a glassy, thready, gray, yellowish, and very tenacious mucus. The glassy, viscid plugs which sometimes fill the tubal openings (so richly provided with glands) simulate those which project from the mouth of the uterus in catarrh of that organ. Masses of mucus often hang down, like stalactites from the roof of the pharynx. The acinous glands are also frequently dilated and prominent, especially in the vicinity of the orifices of the Eustachian tubes.

The epithelium, for the most part, shows no important changes. In some cases, in individual localities, or in greater extent, there is a grayish white tinge, dependent upon an increased thickness of the epithelium in consequence of more copious formation, impeded desquamation, or epithelial suppuration. In the latter case, or upon removal of the epithelium by serous effusion, small *erosions* sometimes occur.

Clinical Remarks.—The milder cases seem to progress without any prominent manifestations. In cases of other severe diseases, local disturbances of moderate severity may be readily overlooked by the patient and by the physician. Respiration through the nose is impeded only in cases of very considerable swelling. The patient then breathes through the mouth, and complains of the constant dryness in the mouth and in the lower pharynx. The voice is altered. These symptoms, however, have but a limited value in the presence of simultaneous affections of the nose. Participation in the disease on the part of the middle ear may occur in various manners (pain, increased by movements of deglutition; hardness of hearing; subjective sensations of hearing). In the few uncomplicated cases which I have observed, there was no fever; on the other hand, there was dulness and heaviness of the head, and occipital pains in the early days, very annoying at the commencement. In the further course of the disease, there is usually a profuse secretion of mucus which hangs down in part from the nares and the posterior surface of the soft palate, and in part upon the posterior pharyngeal wall, exciting the patient to frequent hawking and gagging. The mildest cases last but a few days; the severer cases, those associated with pronounced swelling, continue for several weeks.

Recovery, retrogressive return of the parts to a normal condition, follows either in a complete manner, or alterations remain, mostly of little significance, such as modifications in the form and superficial configuration of the cytogenetic layer, as already described; or the affection passes over into the chronic form, with its subsequent conditions. The aural affections, also, as far as they are present, may undergo recovery in conjunction with that of the catarrh of the naso-pharyngeal cavities. But their highest point may be reached later than that of the latter affection. Not infrequently, secondary diseases remain behind, either of the tube alone, or of the tympanic cavity also, the latter being either secondary (rarefaction of the air), or, as in the case of a catarrh of the middle ear, or of a purulent inflammation ("discharge from the ears"), propagated by extension.

Treatment can be directed only to amelioration of the symptoms especially prominent. In pain, the introduction of very warm fluids (solution of table salt, diluted milk) into the naso-pharyngeal space may be employed by snuffing them up through the nostrils, or they may be applied by means of the nasal douche. At a later period, when there is copious and annoying secretion of mucus, the secretions may be removed by the nasal douche, and by gargling with indifferent fluids (solution of table salt, blood-warm). Astringent influences are injurious here, at the commencement; but they may be employed with advantage, on the contrary, at a later period of the affection, and when it passes into the chronic form (which see).

Complications on the part of the neighboring mucous membranes (nose, ear, lower pharynx) are to be treated accordingly. The ear, especially, often requires immediate *timely* attention, which is of more value than any subsequent course of treatment. The use of the catheter is to be avoided as long as acute manifestations exist. In many cases the methodic removal of the secretions from the naso-pharyngeal cavity suffices also to keep the Eustachian tube free from secretion and render it again a drainage canal and ventilating tube; and the injection of air by the method of Politzer, at stated intervals (daily, at the commencement), is judicious as long as, in consequence of occlusion of the tubes, the atmospheric pressure in the tympanic cavity is different from that outside. In other cases procedures may be necessary, the selection and execution of which require the skill of the specialist (incision of the membrane of the tympanum, among other things).

Chronic Retro-nasal Catarrh.

The naso-pharyngeal space is often found in a condition of chronic catarrh, both in the living and in the dead subject; not infrequently alone, but more frequently in connection with the same or a similar condition of the nose or the lower pharynx, or of both these structures, and also of the middle ear.

Chronic catarrh sometimes proceeds from recurrences of the acute form, or it immediately follows the acute form. For example, in many cases of acute exanthematous disease, a chronic catarrh is developed directly from the acute form, while after "catching cold" several acute recurrences are necessary to establish a chronic catarrh. Or there may be a gradual production of the disease from injurious influences, trifling in themselves, but of continued operation. This is demonstrable in the prolonged respiration of an atmosphere in which irritating particles are suspended, such as smoke, dust, and the chemical (chromic acid, its salts, and the like) or mechanical (flour, fragments of metal and stone, hair, etc.) irritating particles that are produced so plentifully in the processes of various industrial occupations. Or it may become developed under the repeated or uninterrupted influence of unfavorable atmospheric conditions (cold, saturation of clothing, residence in moist, cold localities), and therefore principally in laborers in the open air, in seafaring persons, in the population of the north coast, in porters, in the inhabitants of cellars and other subterranean dwellings.

Allusion has already been made to the frequent appearance of chronic retro-nasal catarrh in connection with prolonged and oft-repeated disturbances of circulation. Of all the various causes, venous engorgement, more than any other, leads to very pronounced alterations.

Finally, unfavorable constitutional conditions must be indicated as causes of chronic catarrh of this locality ; above all, tuberculosis, scrofulosis, and syphilis.

Anatomical alterations.—Hyperæmia is always present in different grades. It is partly dependent upon the amount of blood the individual possesses, upon other diseases with which he may be affected (valvular disease of the heart, emphysema, etc.), and upon the character of the injurious influences at the seat of the disease (as excessive drinking). The hyperæmic tissues exhibit a dirty grayish red, or a dusky dark red color in various shades, a bluish tint in venous congestion, and a dirty copper color in drinkers. In conditions of atrophy, the coarser veins sometimes become very distinctly prominent, tortuous, and dilated, especially around the tubal openings and the choanæ. Small hemorrhages, also, or the remains of them, are frequently encountered in the substance of the mucous membrane in chronic catarrh, especially in congestive hyperæmia, and in diseases disposing to rupture of the vessels, such as atheromatosis ; but it also occurs even under the most varying conditions. An escape of blood may also take place at different points in the floors of ulcers, and about their circumference, and deposits of hæmatoidin in cicatrices are not unfrequent. In consequence of frequent small bleedings upon the surface, the free mucus becomes correspondingly discolored. If the hemorrhages take place in the lacunæ or in the cavernous recesses formed from them, the blood may first stagnate there, undergo the customary transformation, and be discharged accidentally by movements of gagging, or by the introduction of instruments (as the Eustachian catheter). In a case reported by von Troeltsch, which properly belongs here, masses were discharged, after catheterization of the tube, which from their rusty color were at first mistaken for pneumonic sputa.

Thickening of the mucous membrane takes place, in most instances, during the course of chronic catarrh; and in other cases the membrane undergoes thinning. The increase of volume results, in the first place, from dilatation of the vessels, then from a copious serous saturation (especially in congestive catarrh), and then, in most instances, and in many cases principally or exclusively, from an augmentation of the normal or modified tissues of the parts (hypertrophy). Such pronounced deviations from the normal dimensions of the parts as occur in acute catarrh, occur but seldom in the chronic form of the disease. The thickening is usually moderate (the pharyngeal tonsil seldom exceeds a thickness of one centimetre—four-tenths of an inch). The frequent enlargement of the follicles here observed, whose superficial position in the parts, especially at the tubal prominences, imparts the characteristic granular aspect already described (*chronic follicular*, *granular retro-nasal catarrh*), is usually produced by hypertrophic processes in the glands themselves, and sometimes by their suppuration.

Thinning, shrinking (atrophy) of the mucous membrane takes place not infrequently, after long continuance of chronic catarrh. In elderly people retro-nasal catarrh is not often observed in connection with swelling or with hypertrophy of the parts, while rarefaction of the mucous membrane is of very frequent occurrence in such individuals.

Dilatation and hypertrophy of the mucous glands often occur, as a result of which prominences are sometimes visible upon the surface. The secretion is usually very materially increased. The symptoms connected therewith often constitute the chief annoyances of the patients. The surface of the parts is, as a rule, overlaid with a layer of mucus, of varying, often very great thickness, and of a glassy, clear or gray, whitish gray, yellowish white, yellowish or yellowish green color, in most instances of immoderate toughness. Such secretion is not infrequently seen descending upon the posterior pharyngeal wall, or is observed (by means of the mirror) hanging down from the vault of the pharynx, or from the orifices of the Eustachian tubes. The dense, leathery consistence which these masses may acquire (by evaporation) is well known. Such plugs are not infrequently drawn out upon the catheter, adhering with great tenacity to its beak.

This locality is very frequently the seat of cyst-like formations in cases of chronic catarrh; and even true cysts are occasionally developed here. Concerning their origin (usually from parts of the lacunæ), their contents, their often remarkable size, and their numbers, see what has already been written.

The following condition, which is observable in many cases, is characteristic of retro-nasal catarrh associated with hypersecretion. On the pharyngeal tonsil, on the tubal prominences, and in the fossa of Rosenmueller, we see numerous fine apertures, and somewhat larger round and also slit-shaped openings. Even moderate pressure will force numerous isolated small drops of mucus from the former, and from the latter lumps of mucus and colloid material of varying and sometimes colossal size, often in great numbers. The former are orifices of distended mucous glands; the latter are cystoid dilated portions of the lacunæ or tubular glands, the result of pathological processes. (In a similar manner, such hollow spaces may be evacuated during digital exploration, or through introduction of the catheter, in consequence of the pressure exerted in these manipulations.) In many cases closed cavities remain, the contents of which glisten through with a gray or yellowish gray lustre; and they may even push the surface forward—true cysts, unchangeable by pressure, but readily torn, on the other hand, by means of the catheter. (Such occurrences are mentioned by von Troeltsch.)

The epithelium is here thickened in individual cases, and assumes a whitish gray color, especially on the posterior wall. I have also sometimes seen, in and near the edges of ulcers, a proliferation of epithelium (with or without a deposit of pus) and its elevation by newly-formed papillæ.

I find erosions and small superficial losses of substance not altogether infrequently at various points ; ulcers of greater depth and extent (up to a diameter of six millimetres—one-quarter of an inch), circular, with steep thickened edges, occur in a few cases only, and almost solely where the posterior wall merges into the pharyngeal tonsil, or rarely in the very substance of the tonsil. Here we often find small follicular ulcers, singly or in groups, but of less regular outline.

Symptomatology.—The symptoms of chronic retro-nasal catarrh are chiefly dependent upon the character and quantity of the pathological secretion, and the amount of augmentation in thickness of the mucous membrane. An actual occlusion of the choanæ by the swollen or hypertrophied pendent pharyngeal tonsil occurs only exceptionally. More frequently the portion remaining free is periodically filled up with tenacious masses of secretion, as is shown by a change in the symptoms (continuous breathing through the mouth and altered speech), as well by the results following the successful use of the nasal douche. The swelling at the orifices of the Eustachian tubes (which are notoriously wide) is seldom so considerable as in itself to produce an occlusion of the middle ear with its immediate mechanical results (rarefaction of the air). But even a moderate constriction favors the complete abolition of the calibre of the tubes by viscid mucus. Such a condition occurs in a certain ratio of cases, even without marked swelling. In cases of great increase of thickness of the mucous membrane, the muscles of the palate may become relatively insufficient, and in case of their atrophy, absolutely so (von Troeltsch). Difficulty of swallowing does not readily occur under such circumstances ; but there is an insufficient ventilation of the middle ear, if the contractions are not sufficiently powerful to render the orifice of the Eustachian tubes patent.

The presence of a great amount of secretion, especially when of dense, tenacious, leathery consistence, sometimes produces an annoying sensation of heaviness and confusion in the head, or the sensation as of the presence of a foreign body. Headache also occurs now and then, fleeting, or of longer duration. Many patients feel themselves driven to characteristic movements of the soft palate in order to draw the mucus down, or to frequent hawking when it has descended into the lower pharynx (see above). With very slight secretion (in atrophic conditions) there is often a feeling of dryness ; and so, likewise, when any considerable portions of the nose are destroyed.

Concerning the manifestations in simultaneous disease of the nose, the lower pharynx, and the ear, consult the appropriate sections.

All the symptoms, those arising from the naso-pharyngeal cavity itself, as well as those from the contiguous organs, are usually more prominent at the close of the night's rest, during which the secretion has not been removed at all, or has not been sufficiently removed. They are increased, further, by whatever augments the flow of blood to the parts (rich food at meals, the use of spirituous liquors), or impedes its reflux in the veins (continuous stooping, tight cravats, the knapsack and clothing of soldiers, etc.).

Not a few cases progress almost without symptoms. Often it is the consecutive affection of the ear which brings the patient to the physician. Affections of the ear occur here much more frequently, and are more often extended over the entire middle ear (chronic catarrh, also suppuration), than in acute retro-nasal catarrhs ; and, not infrequently, they lead in the course of time to the highest grades of functional disturbance.

Even inveterate catarrh of this region is curable under favorable constitutional conditions, or after the improvement of unfavorable conditions, by the institution of suitable treatment with a proper amount of co-operation on the part of the patient. In many cases improvement only can be accomplished, and that to a variable and often to but a slight extent. This is true in cases in which other important local or general diseases persist, in cases occurring during advanced life, or in case of the continued action of injurious influences (as in habitual drunkards and persons following certain avocations). Sometimes the only result that can be obtained is the warding off of annoying symptoms by means of some simple procedures, to be employed daily by the patient himself.

Recurrences after recovery, or exacerbation of existing processes, especially as a result of "catching cold" (every winter, therefore, in predisposed individuals), and in consequence of general or local diseases (in the nose or lower pharynx), are not infrequent.

When left to itself, retro-nasal catarrh is of indefinite duration. At first, hypertrophic conditions are frequently developed. The final termination after many years' duration, in advanced age, is atrophy.

The aim of the local *treatment* is to secure careful removal of the secretions, decrease in the amount of secretion, and cure of the swollen conditions.

The nasal douche and gargling, both of especial importance early, are to be employed several times a day; the former with a five-grain solution of table salt, or with a solution of bicarbonate of soda of the same strength, or with a mixture of the two. Their employment leads to an improved condition of the cavities, which usually takes place soon; but in cases of great viscidity and copiousness of the secretion, only after prolonged use, even continued for months. The cavities are freed from the coarser masses, and by continuing the operation, they are always kept approximately clean, and in this way the annoying symptoms subside. Recovery, also, is promoted by their use, or, at any rate, a condition of the mucous membrane is reached which renders it possible to act upon it medically, as is not the case when it remains covered with a layer of stagnant and closely adherent secretion.

In this connection the daily washing of the parts once or several times, with an astringent, by means of the nasal douche—always after careful cleansing with salt water—comes in question. The best preparation for this purpose is a solution of sulphate of zinc, half a grain to the ounce. The remedy in this weak dilution, which should not be exceeded without caution, produces a slightly irritant, astringent influence upon the mucous membrane of the nose and the naso-pharyngeal cavity. Stronger solutions are rarely borne (they produce headache, burning in the nose, irritation of the conjunctiva). With other remedies (tannin, etc.) I have had less success. I caution against the use of alum in a fluid form, because I have seen permanent loss of smell after its use, in three cases.

If we desire occasionally to introduce more concentrated solutions (nitrate of silver, from fifteen to fifty grains to the ounce, sulphate of zinc from fifteen to twenty-five grains) into the naso-pharyngeal space, avoiding the irritable nasal passages, we may saturate a sponge on the end of a probang with the desired fluid, and pass it up through the pharynx. If we wish to employ a long hair pencil through the healthy nose, the concentration of the solution must be decreased; at least, it should not exceed the lower limit of the proportions given above. This precaution may often be unnecessary in chronic diseases of the nose, which endow the parts with greater tolerance and even require the employment of stronger remedies. Injections through the pharyngeal catheter, from the nose, must be practised only with such medicaments as may gain access to the stomach without injurious consequences.

The frequent (once or twice a week, daily at first) insufflation of powders is often very useful, tried at first, and continued according to the indications. The best powder is very finely pulverized alum (pure, or in a mixture with equal parts of gum arabic), blown through a tube passed through the lower pharynx, or through the nose (see general treatment). These powders act stimulatingly and astringently upon the mucous membrane, and are very well borne for the most part. Carbonate of soda, common salt, a mixture of both, or sal-ammoniac, may be employed in this way with great service, in cases of the retention of very viscid and adherent masses of secretion. In very obdurate cases, in cases of especially copious collections of mucus, and severe swelling, it is good practice to use, once or oftener, a powder composed of one part of nitrate of silver with from eight to ten parts of a mixture of gum arabic and sugar. This usually excites at once an increased but thin fluid secretion, often sneezing and burning, and sometimes headache also. The extrication of masses by gargling, gagging, and by the nasal douche, takes place more readily. After the first storm is over, the patient feels a remarkable alleviation (in many cases, in relation to the ear also), which, however, is not often of long continuance at first.

The cauterization of hypertrophic parts with nitrate of silver in substance, is practised through the nose or through the lower pharynx, by means of sounds or probes which carry a small globule of the material on their extremities. Those to be introduced through the nose are protected by tubes. Concerning these points, and also concerning the use of the galvano-cautery, and other operations, see under Hyperplastic Catarrh and Morbid Growths.

The treatment of the diseases of the nose and the lower pharynx, so often present, is not to be omitted (see the sections in question). The middle ear, so often involved, demands, above all, *careful* attention on account of the danger to its functions. Although the improvement of the retro-nasal catarrh often exerts a beneficial influence upon the associated aural affection, still, certain conditions of this organ occur during its continuance which require intelligent special treatment (use of the catheter for various purposes, paracentesis of the tympanic cavity, etc.). The simple procedures recommended in treating of acute retro-nasal catarrh, are generally not sufficient here. If, on the contrary, these conditions are neglected, they are very likely to result in chronic forms of disease, of indefinite duration, and very intractable to subsequent treatment. Such forms, being subject to exacerbations upon very slight provocation, and at least growing worse every winter, often lead, early, to a severe grade of deafness in consequence of secondary changes in the mucous membrane (sclerosis, calcareous deposits, etc., and thus to immobilization of the sound-conducting apparatus), and less frequently to deafness in consequence of destructive processes.

Besides the local treatment, constitutional treatment is essential in the case of many patients, especially in those who are syphilitic, scrofulous, tuberculous, or anæmic. Residence at a well-selected watering-place may be of important service.

In certain cases of old inveterate catarrh, which had withstood every therapeutic measure, and in which there was no evidence of syphilitic infection, I have seen improvement after the protracted use of iodide of potassium, and especially under the influence of calomel (four grains at a dose given daily, and continued from ten to fourteen days, repeated several times if necessary).

To prevent recurrences in cases that have been cured, and exacerbations in cases of existing retro-nasal catarrh, avoidance of the injurious causal influences, and methodic hardening of the skin, are not to be dispensed with.

Acute and Chronic Catarrhal Inflammation of the Lower Pharynx; Acute and Chronic Pharyngeal Catarrh.

Acute Catarrh of the Lower Pharynx.

This seldom occurs by itself. The larynx, the mouth, or the naso-pharyngeal cavity is often diseased at the same time. The soft palate is the part most likely to participate in the disease. This is usually the part principally affected and furnishing the most important symptoms, while the affection in the lower pharynx appears much less severe. Thus, there is sometimes simple catarrh of the latter in association with phlegmonous inflammation of the palate.

The same injurious influences occasion acute catarrh of the palate and of the lower pharynx (atmospheric influences, chemical and mechanical irritation from ingesta, overstraining of the parts); the same acute and chronic diseases are likely to be accompanied by affection of these parts simultaneously. An acute catarrh of the lower pharynx may also occur without angina, alone or associated with catarrh in the larynx, especially after "taking cold," after excessive smoking and drinking, after too much singing and protracted speaking. We must attribute a certain influence to individual predisposition in accounting for the fact that sometimes the one and sometimes the other of the organs affected in common by the same injurious influences, will be prominently or solely diseased.

The *anatomical changes* are often less marked in the lower pharynx than in the palate. The redness is uniformly less; it is diffuse or attended by prominence of some venous plexus. The swelling only exceptionally reaches a severe degree; it is uniform or presents small flattish prominences on the surface (dilated mucous glands, swollen follicles). Sometimes there is also a formation of vesicles, of excoriations, or of small follicular ulcers.

Mild cases progress without any special discomfort. In severe cases the local *symptoms*, at the commencement, are heat, dryness, sensation of tension, and difficulty in swallowing, but pain is infrequent; and at a later date there is a copious secretion of mucus (causing frequent hawking, gagging, even vomiting, and cough).

When the entire pharynx participates, all the general symptoms of angina may be increased. Fever is seldom present in simple catarrh of the lower pharynx, but headache is more frequent.

Course.—In cases of catarrh limited to the lower pharynx, recovery often follows in a few days. In other cases it requires a period of from one to two weeks, seldom longer. Sometimes it passes into the chronic form of the disease. In very infrequent cases there is a fatal termination by extension of the process into the larynx (from œdema of the glottis). Such cases have been reported by Rilliet and Barthez, Bamberger, Ruehle.

Treatment.—In many cases there is no necessity for any special treatment. As a matter of course, certain injurious influences (irritating ingesta, smoking, etc.) must be abandoned. In case of pain, and in severe cases generally, cataplasms, moist and warm applications around the neck, may be employed with advantage, and so, also, derivatives to the skin (sinapisms, foot-baths) and to the bowels. Locally, advantage will be derived from frequently repeated inhalations of simple warm vapor of water or of salt water, or a solution of muriate of ammonia, or the pharyngeal bath may be used with a decoction of mallow or the like; and, in the absence of inflammatory pain, gargles with a solution of chlorate of potash. (For severe forms of the disease, see under treatment of phlegmonous inflammation). To prevent recurrences, systematic hardening of the surface of the body in general, and of the skin of the neck in particular, and the avoidance of the ordinary injurious influences (smoking, drinking, unnecessary breathing through the open mouth, too great exertion in speaking and singing) are to be recommended.

Chronic Catarrh of the Lower Pharynx.

This supervenes upon acute catarrh as a direct sequel, or in connection with mild recurrences due to slight exposure. It may also gradually ensue under the influence of the repeated or the continuous operation of injurious influences (see acute catarrh), or in consequence of disturbances in the circulation (in heart and lung affections, etc.). It occurs by itself more frequently than the acute form, and it may occur with simultaneous catarrh of the larynx or the naso-pharyngeal cavity, or with angina.

Anatomical Changes.—In most instances there is hyperæmia, which may exist in very various grades. It is especially pronounced in drinkers, and also in cases of venous stasis, always modified according to the quantity of blood in the patient. Extensive dilatation readily occurs, and varicose dilatation too, with contortion of the coarser veins, here so numerous in the deeper layers. There is not often any very considerable increase of thickness in the parietes, even when the hypertrophy is marked. On the other hand, the appearance of individual or numerous flat prominences, uniformly colored, or from a gray to a reddish gray color (swollen and newly formed follicles ; dilated and hypertrophied mucous glands), is a very common thing in chronic catarrh of the lower pharynx—a condition which is then commonly called *pharyngitis granulosa.* In the course of this affection conditions of atrophy may also become developed.

The squamous epithelium on the posterior pharyngeal wall in some cases undergoes a greater increase at individual places or over a certain extent, so that there may be an appearance as of a grayish white layer, or even a pseudo-membrane. Roundish ulcers are infrequent, and do not readily acquire any great extent or great depth. They represent destroyed solitary or grouped follicles.

Symptoms of importance are often absent. When such are present they are generally of the nature of various subjective sensations which are principally dependent upon increased or diminished secretion—sensations of dryness, burning, rawness, disposition to hawk, cough, etc. Smokers and drinkers do not complain much of these annoyances, which are incident to their habits. These manifestations are most evident generally in the mornings or after over-exertion (in teachers and singers), or after excesses.

Severe grades of catarrh are often associated with slight local symptoms. In some persons, on the contrary, especially in hysterical individuals, there is a great irritability of the mucous membrane, a regular hyperæsthesia under the most insignificant influences, although the changes of structure are very moderate.

In some patients catarrh of the lower pharynx gains more importance from participation of the larynx (hoarseness, cough, loss of voice) and of the naso-pharyngeal space, especially the tubal orifices (hardness of hearing, noises in the ears).

Course.—Whether the chronic form of the disease is cured, or whether it continues for years, to subside finally in atrophy, depends chiefly upon the causes which have given rise to it and have maintained it. If it is not practicable to avoid the injurious influences, to cure or improve the constitutional and important local affections, then, as a rule, no permanent relief to the pharyngeal catarrh is to be anticipated from local remedies. On the other hand, it is usually practicable even then to effect an amelioration as far as the symptoms are concerned.

Treatment.—Local treatment may be employed, in the form of gargling, to remove the secretions, more especially in the morning. To produce an astringent influence upon the mucous membrane, we may resort to frequent gargling daily and the use of the pharyngeal bath, and to inhalations of alum, tannin, etc.; to pencilling the accessible parts, once or twice a week, or even daily, with solutions of nitrate of silver or of sulphate of zinc (from fifteen to thirty grains to the ounce). Other measures are requisite in conditions of atrophy or hypertrophy of the mucous membrane (see farther on).

The general treatment, which is often important, and must always be associated with the local treatment, is to be instituted according to the conditions of the individual case. The choice of a bathing-place may be especially governed by individual requirements. It is not every one that will be cured or improved at Ems; more patients are to be recommended to visit Aix-la-Chapelle, Marienbad, or Kreuznach.

Phlegmonous Inflammation of the Naso-pharyngeal Cavity; Retro-nasal Phlegmon.

This affection, associated with severe swelling and hypertrophy, with suppuration of the parenchyma, and with pain, occurs after operative procedures in the naso-pharyngeal space, especially after the use of the hot iron and after severe cauterization in the removal of morbid growths; and it may then extend to the lower pharynx also.

In one case, in which death took place on the fifth day after resection of a carcinomatous upper jaw, I found sero-purulent infiltration (with hemorrhages) in the pharyngeal tonsil, at the orifices and prominences of the Eustachian tubes, and in the levator and tensor muscles; pure purulent infiltration, with abscess, in some places; and ulceration at one tubal orifice. In two peculiar cases (in 1871) I saw reactive inflammation of the naso-pharyngeal cavity, which was attended with severe pains and the secretion of purulent mucus, and which led to inflammation of the middle ear, after removal of portions of the pharyngeal tonsil with Meyer's ring (see below). After five or six days the old condition had returned, excepting as regards the ear.

An anæmic young man, seventeen years of age, whose ears were sound, and whose hearing was excellent, but who could breathe only through the mouth, and spoke without use of the nose, sought relief on that account. On examination by the finger and the mirror, the pharyngeal tonsil was found to extend far downwards, to be hypertrophic, and to be divided longitudinally and lobulated. It was removed by me with Meyer's ring, in several sittings of long duration. The expected improvement duly followed; but at the close of the last operation a reaction set in, associated with headache and with severe pains in the right ear. Repeated paracentesis of the affected tympanic cavity became necessary. It was a long time (a quarter of a year) before the sero-purulent inflammation of this cavity was fully healed. The tolerance of the patient permitted the freeing of the vomer and the choanæ, and the healing of the surface of the wound, to be watched from time to time by means of the mirror.

In a scrofulous girl, twenty-two years of age (with mucous catarrh of both middle ears, only temporarily benefited by the usual remedies, and with oral respiration, and altered speech), there was a remarkable increase of thickness in the pharyngeal tonsil, in addition to hypertrophy of the palatine tonsils, which latter I at once excised. The former presented to rhinoscopic and digital exploration a lobulated and granular surface. After repeated use of the ring, there was headache, intense pain in both ears, and spontaneous rupture of one tympanic membrane. Considerable alleviation followed leeching, the use of cathartics, etc.

Wreden narrates a case which, from its origin and its course, is characterized as phlegmonous. Caustic liquor ammonia had been inadvertently poured into the nose of an eclamptic woman so that the caustic fluid penetrated into the naso-pharyngeal space, and into the Eustachian tube and tympanic cavity of one side. Very severe pains were immediately felt in the nose, pharynx, and ear, and continued until the end of the third day. Large white flakes were discharged from the pharynx and from the nose.

I also include, in this category, those cases in which, during the course of small-pox, in addition to severe swelling of the parenchyma, there is epithelial suppuration in the naso-pharyngeal cavity (on the tonsil, and almost equally often at the orifices and prominences of the Eustachian tube, but almost never in the tube itself). According to my own observation, this takes place in more than half the cases. It proceeds to the formation of small insulated or larger membranous, soft, yellowish deposits in the place of the epithelium, in consequence of a change in it (increase and distention of the cells, with purulent infiltration in and between them). The deposit is soon thrown off. According to the extent it has reached, there remains a (shallow) ulcer; or the entire surface is raw, as though excoriated. There is, with it, hemorrhagic and sero-purulent infiltration of the connective tissue of the anterior region of the tube, and of the cytogenetic layer; and the latter may acquire a carious friable consistence and a discolored appearance. Retrogression appears to take place rapidly; it does not progress to the formation of cicatrices. Destructions and adhesions of parts in the naso-pharyngeal space should not be attributed to the small-pox, as was done by Lindenbaum in regard to a case of closure of the orifice of the Eustachian tube by a cicatricial membrane, the tubal prominence also having been destroyed.

The specific process in variola extends to the tympanic cavity only in very rare instances (grayish efflorescence, epithelial suppuration). On the other hand, I have frequently found a high grade of hyperæmia here (also in the form of hemorrhagic catarrh of the cavity, accompanying variola) often associated with hypersecretion, suppuration, and swelling. Only once have I seen a diffuse variolous inflammation of the dermoid layer of the membrana tympani with the formation of vacuoles.

The treatment of phlegmonous inflammation in this locality, aside from derivatives, can only consist in the careful removal of the secretions, the ichor, and the pus. Where the ear is simultaneously involved, the use of the catheter is as a matter of course out of the question.

Phlegmonous Inflammation of the Lower Pharynx; Pharyngeal Phlegmon.

Phlegmon of the lower pharynx represents to a certain degree a higher grade of catarrhal inflammation. A middle type is represented by forms of inflammation which are intermediate between these extremes. Phlegmon takes its origin in the lower pharynx, less frequently from a general "cold," or from chilling of the throat or the feet, than from direct toxic (mineral acids, caustic lye, etc.) and thermic (scalds) injuries to the mucous membrane, and from operative interference. In some severe affections (typhoid fever, acute exanthems) there is a phlegmonous inflammation of the lower pharynx, mostly of a mild type. The palate is also similarly diseased, as a rule. Not unfrequently there is, at the same time, catarrh of the nose, the naso-pharyngeal cavity, the middle ear, and the larynx.

Anatomical Alterations.—The mucous membrane of the lower pharynx is intensely congested, often bestrewn with hemorrhages, and infiltrated with serum. With this there is a more or less rich infiltration with pus cells. It rarely proceeds to the formation of purulent collections.

The local *symptoms* which the milder forms of phlegmon occasion are similar to those in severe forms of acute catarrh: at first a sensation of dryness, and, later, copious secretion of mucus and also of pus. There are burning and stinging pains, spontaneous, or upon contact and movement, especially in efforts at swallowing, and also upon movements of the head. In cases of very severe swelling, nothing at all or nothing but fluid can be swallowed. Sometimes there is a foul odor from the mouth, and always nausea and disposition to vomit, and these also are produced by the local trouble (Quarin, Ruehle). Speech, even in mild cases often altered and difficult, becomes impossible. Dyspnœa may be produced and may even progress to suffocative paroxysms, which may render tracheotomy necessary.

The phlegmon is usually attended by general symptoms: fever, languor, headache, thirst, loss of appetite, and sleeplessness. According to Bamberger, whose excellent description I follow here, delirium is sometimes present. He himself saw maniacal paroxysms in two cases. The intensity of the fever and the prominence of the local symptoms are especially influenced by the grade which the affection reaches, and the method of its production; the worst forms are those produced by thermic influences.

Course.—Sometimes the phlegmon begins with a general feeling of illness, rigors, or even a chill, occurring for the most part at the same time with the local symptoms, which increase during the first few days, and decrease again after a few days more.

Recovery may take place in from one to two weeks, in favorable cases. In severe forms the course of the disease may be very protracted. A fatal termination has been observed occasionally, in some cases following the removal of tumors, by the production of œdema of the glottis or of pneumonia. In one such case (Stroppa) the œsophagus was inflamed as well as the larynx, and there was extensive suppuration of the connective tissue of the neck. Bamberger considers a termination in gangrenous destruction of the affected parts as very infrequent, and liable to take place only in very reduced patients under highly unfavorable external conditions.

Recovery is either complete, or the affection eventuates in chronic catarrh, or it may terminate in hypertrophy or atrophy of the mucous membrane.

With reference to the *diagnosis*, that rests principally upon the nature of the causal injuries, as well as upon the pain, which otherwise is not likely to occur in acute affections of the lower pharynx.

Treatment.—In the milder forms the treatment recommended for acute catarrh is sufficient. In severe cases the principal indication is to abate the inflammatory swelling of the mucous membrane. This is attempted by the employment of cold (bits of ice in the mouth, iced drinks, ice-bladders around the neck), the abstraction of blood (leeches behind the angle of lower jaw), derivative procedures (cathartics, foot-baths, etc.), and scarification (in cases of especially severe swelling).

In England, where severe phlegmon frequently occurs in little children from scalding with hot water (drinking out of the spout of the tea-kettle), tracheotomy is often necessary. Bevan reports twenty-five cases of death in thirty-six operations; and he recommends calomel, four and a half grains, every half hour. Improvement is said to set in with the appearance of calomel stools, after eight, or at most after from eighteen to twenty-four hours. (Bamberger especially advises against the use of calomel.)

Cauterization with nitrate of silver in substance, the insufflation of powdered alum, or its direct local application, and the use of stimulating and astringent applications generally to the inflamed parts, are recommended from various sources. Their employment is attended with inconvenience and pain to the patient; and opinions are divided as to their utility. Emetics are occasionally employed (Bamberger) to remove adherent secretions, or in cases of œdema of the glottis; but are otherwise of no use, and they increase the pain.

Pharyngeal and Retro-Pharyngeal Abscesses.

Abscesses of the Lower Pharynx.

Small isolated collections of pus, reaching the dimensions of a pea, sometimes occur in the wall of the *lower pharynx*, by suppuration of follicles, by confluence, and by breaking down of the intermediate tissue. They are larger in phlegmonous inflammation, but even here do not readily reach any considerable extent. They are manifested by their yellowish color, and also, in case of the larger abscesses, by the prominence and fluctuation of the affected spots.

By spontaneous rupture during sleep the pus may descend into the air-passages and produce suffocation. If there be any delay in opening the abscess, there may be a descent of the pus (in a case of Londe as far as the thoracic cavity with a fatal termination). It is therefore essential that they should be timely incised, if the collection of pus is at all copious.

In the *naso-pharyngeal cavity* I have seen small abscesses within the cytogenetic layer only in isolated cases. They occurred in phlegmon (see above), or they were produced by the destruction of many contiguous follicles (among other things, with simultaneous suppuration of the palatal tonsil). They may be termed *naso-pharyngeal abscesses.*

Retro-Pharyngeal Abscesses.

The abscesses about to be described are not situated in the walls of the pharynx itself, like the pharyngeal abscesses, but outside of it and behind it. They occur chiefly in disease originating in the cervical vertebræ and their ligamentous apparatus (in caries, fracture, especially of the atlas and axis), in the lax, movable connective tissue which is spread out between the vertebræ and the posterior pharyngeal wall, and also in the lymphatic glands there situated. They are of very varying size and extent. They are found as circumscribed tumors, from the size of a pigeon's egg to that of a horse-chestnut (in glandular suppuration, in caries of the vertebræ). They may acquire a very remarkable size. They are then correspondingly extended over the greater portion of the posterior wall, reaching downwards even to the œsophagus (primary suppuration of the connective tissue ; descent of pus from glandular or vertebral abscesses). In some cases there is such a marked prominence of the posterior pharyngeal wall that the palate is pushed forwards.

Most of these abscesses affect the *lower pharynx* and its posterior wall ; when of greater extent, they stretch also to either side. Only those belong to the *naso-pharyngeal space* which originate from the anterior arch of the atlas, from the odontoid process of the axis, or from the lymphatic glands here situated, and remain limited to this region (posterior wall of the cavity). The naso-pharyngeal space may also be reached, and even the vault itself (basilar process) may be reached in acute inflammation of the connective tissue (retro-pharyngeal phlegmon), as in a case examined after death and reported by Abelin. In a case of caries of the occipital and sphenoid bones, I found a collection of pus between them and the pharyngeal tonsil.

Diffuse inflammation of the retro-pharyngeal connective tissue occurs most frequently in children at an early age, and is by no means very infrequent. The formation of retro-pharyngeal abscess is most frequent up to the tenth year of life.

The commencement is often sudden (in acute suppuration of the connective tissue), and the course is rapid, showing symptoms which are like those of phlegmon of severe grade, and are developed in varying intensity in different cases—fever, sleeplessness, dyspnœa, difficulty in deglutition, pain, increased on motion of the head, stiffness in holding the head, spasms in very young children (Abelin), and convulsive paroxysms (Hacker).

In most cases (especially in affections of the vertebræ) the commencement is not marked, and the course is slow. The symptoms are then sometimes less prominent, or certain symptoms only (inability to turn the head, difficulty in deglutition) are present.

The abscess, left to itself, opens spontaneously and discharges its contents into the lower pharynx, or fistulous tracks are formed towards the thoracic cavity, or towards the skin of the neck. In one such case (Halton), after the premature closure of a fistulous opening in the lower portion of the neck, there was a sudden refilling of the cavity of the abscess, with very alarming manifestations, which disappeared on the re-establishment of the discharge. A fatal termination may ensue by suffocation when, on spontaneous rupture, especially during sleep, or when, on opening the abscess with the knife (Gaupp), the pus gains entrance into the air-passages; or from occlusion or compression of the larynx by the enormous size of the tumor; from secondary disease of the larynx and the thoracic organs (among other things, occasioned by descent of pus into the thorax). In a case of retro-pharyngeal abscess observed in the clinic of von Pitha, reported by Scholz, there was thrombosis of the internal carotid artery, the internal jugular vein, and the transverse sinus.

The *prognosis* in general is most unfavorable in abscesses originating from caries of the vertebræ. Here the original disease, which is slow to heal, remains behind (cases of discharge of the odontoid process, etc., after fracture), or there exists the uncomfortable necessity of supporting the head with the hand, or with special contrivances. The inflammatory abscesses of the connective tissue, or the simple lymphatic glandular abscesses may heal without untoward results.

The *diagnosis* is readily made in marked cases by attention to the clinical manifestations, and by visual and digital examination of the diseased parts (a more or less circumscribed fluctuating tumor). The superficial (collateral) œdema of the pharyngeal mucous membrane over the abscess, usually present, also prevents mistaking it for phlegmonous inflammation of the pharynx. Large cystic tumors of the throat, which may cause dysphagia and suffocation (by pressure, or by escape of their contents into the pharynx), are readily recognizable.

The *treatment* can be directed in the main only to the earliest possible opening of the abscess. To prevent the penetration of the pus into the larynx, Abelin and others recommend the use of a trocar, as well as promptly bending the head forwards.

Croupous and Diphtheritic Inflammation of the Naso-Pharyngeal Cavity and the Lower Pharynx.

The disease itself has already received a very thorough discussion in various portions of this work, so that I may confine myself here to its local description.

Fournier describes the occurrence of a diphtheritic affection in the naso-pharyngeal space *only*, or, rather, as his description indicates, affecting the choanæ also, consequently the nose. Five cases came under observation almost at the same time, and progressed favorably.

I have very rarely encountered croup and diphtheritis in the naso-pharyngeal cavity alone, and confined to it; it is equally rare to find them with simultaneous involvement of the nose only when they occur secondarily in chronic tuberculosis or in small-pox. Apart from this, I have seen specific alterations in the naso-pharyngeal space, in almost half the cases in which the palate and lower pharynx, or the larynx, or all these structures together were affected. In the other half of the cases of croup and diphtheritis, in which this cavity remained unaffected, and also in croup of the larynx simply, there was hyperæmia, often of a severe grade, in most instances associated with hemorrhages, and, except in a single instance, with swelling of the cytogenetic layer, especially of the tonsil.

The deposit either occurred only on the vault, or on the lateral walls also. In one such case, where it was not yet detached from its connection with the mucous membrane, it formed a regular cast of the cavity. Its immediate transformation into membranes which covered the posterior surface of the soft palate, or sank into the choanæ, or covered merely the posterior edge of the septum, was seen in several instances. In other cases no direct connection was visible. Its consistence, compared with that of the deposit adherent to the palate and lower pharynx, indicated in part a later, and in part a simultaneous origin.

With this there was, in most instances, intense swelling (serous, cellular and hemorrhagic infiltration), a discolored aspect, a rotten, broken-down consistency. The surface appeared covered with tenacious sanguinolent ichor, and also with blood-clots. The mucous membrane of the Eustachian tubes exhibited but slight changes in those cases in which it participated in the formation of membrane. According to the classification of E. Wagner, it was mostly diphtheritis in the naso-pharyngeal cavity, and always croup in the tube. (See Croup and Diphtheritis; Inflammation of the Soft Palate.)

In most cases the process also extended to the posterior portions of the nose, sometimes also upon both nasal passages in their entire extent. It did not occur in the nose alone.

In a fifth of the whole number of cases of croup and diphtheritis, and in two-fifths of those cases in which the naso-pharyngeal space participated, but in no instance without immediate connection with the corresponding affection of this space, I found an extension of the specific process into the middle ear. (In some other cases the tubal prominences were covered with membrane, which terminated abruptly at the orifices.) There was found, always bilaterally, a tube-shaped croup-membrane, or a solid cast in the cartilaginous tube. In one instance only, the formation of membrane reached the cavities of both middle ears and of both mastoid processes, and even affected the coverings of the ossicles. With the exception of this case, hyperæmia of the mucous membrane, and also hemorrhage, were the only changes observable in the osseous portion of the middle ear. In the remaining cases of croup and diphtheritis (with and without the formation of membrane in the naso-pharyngeal cavity), there was catarrhal, and also purulent inflammation, or simple hyperæmia of this cavity, and in several cases (even with the presence of deposits over the entire tonsil) its condition was perfectly normal.

In the cases of croup and diphtheritis in Leipzig and its environs, especially in the course of scarlatina, acute inflammation of the middle ear in its ordinary form occurs not unfrequently. This gives rise to chronic suppuration of the ear, often the result of inefficient treatment, which in the course of time may give rise to the formation of polypi and the production of caries; thus endangering the functions of the ear and the general health. In earlier epidemics, it was not an uncommon occurrence for processes to take place which rapidly led to destruction of the bones, and to the severest grades of hardness of hearing, even to deafness and deaf-muteism. These, however, must not be regarded as specific, especially as severe pain was always present, and, what is more, suppuration, even at the commencement of the destructive process. Things may be different in other places and in other epidemics. Wreden (Petersburg) reports eighteen cases of nasal and (lower) pharyngeal diphtheritis in scarlatina, in which the ears were very much diseased (both ears in sixteen cases). The rapid production of important changes (destruction) in the parts, without pain, even, also without suppuration, and the course of the disease, indicate a specific affection of the ear transplanted from the naso-pharyngeal space.

When a fatal termination does not ensue from the general disease, or from the disease of other organs, recovery takes place; or a chronic retro-nasal catarrh remains behind. (Concerning paralysis of the muscles of the palate and Eustachian tubes, and the resulting disturbances in hearing, consult the section on the Neuroses.)

The local *symptoms* are chiefly caused by narrowing or occlusion of the calibre of the cavity, and are still more pronounced when the nasal passages are involved (modification of speech, of respiration). Then there is sometimes difficulty in swallowing, and, rarely, pain, almost always only in the ear. General symptoms (fever, depression, confusion of ideas, headache) are present in different grades, according to the individual cases. The fever especially (and often the headache also) undergoes a diagnostically valuable increase, in cases of participation of the naso-pharyngeal cavity, and in simple inflammation of the ear or exacerbation of existing inflammation. In the cases of almost exclusive disease of the naso-pharyngeal space, reported by Fournier, there was slight chilliness (at the commencement), lassitude, headache, nausea, hot dry skin, and the local symptoms already detailed.

The *diagnosis* is founded on the appearance of severe local and general symptoms with the absence of a corresponding amount of pain or its total absence, and by the presence or discharge of membranes.

The *treatment* has for its object the removal of the membranes, ichor, and secretions from the cavity, as well as favoring the recession of morbid processes in the mucous membrane. These are facilitated by injections through the nose, and by the use of the nasal douche, which for the most part is well borne, and affords considerable relief to the patient. Indifferent solutions are employed for this purpose, or solutions of permanganate of potassa, carbolic acid, and, at a later period, astringents. In addition, therapeutic measures, directed against the general manifestations of the disease, are necessary in some cases. The diseases of the ear that occur in this connection require careful and timely treatment; and the measures instituted to this end must be judiciously selected and carried out (abstraction of blood, cataplasms, filling the external ear with warm water, incision of the tympanic membrane, timely and frequent use of the syringe, the inflation of the middle ear with air by Politzer's method or by the passive expedient of Valsalva). The existence of aural affections, which are not always evinced by discharge and other unmistakable symptoms, renders it advisable that care be used in these cases, as in other acute affections, in the employment of cold baths and sponging, and also in the application of ice and cold compresses to the head, a caution which may be advisable under such conditions, even in cases in which the ear is intact. The penetration of cold water into the ear, which always injures a diseased ear, and often a healthy one, must be prevented by the introduction into the meatus of a firm wad of charpie or cotton, judiciously saturated with oil or grease.

The characteristic deposits, without strongly marked alterations in the mucous membrane—croup, or with them diphtheritis—seldom occur alone in the *lower pharynx*. On the other hand, this region is often affected at the same time with the palate, and also with the larynx or the naso-pharyngeal cavity. The formation of membranes takes place most frequently on the posterior wall, and they are present in isolated patches or in greater extent. The deposit sometimes appears earlier in this locality than on the palate and other localities, or it may appear subsequently.

The *symptoms*, especially the fever and the difficulty of swallowing, are the same as in croupous and diphtheritic angina.

The diagnosis is facilitated by the readiness with which the part may be submitted to visual examination. The *treatment*, in addition to attention to the general condition and the casual complications on the part of other organs, the larynx especially, may consist merely in removal of secretion, the repression of disagreeable odor, etc., by injections of weak solutions of carbolic acid or lime water, or the frequent slow swallowing of a solution of chlorate of potash or some other suitable fluid uninjurious to the stomach, or the inhalation of such solutions. Leeches, and ice compresses have little beneficial action here, though they are somewhat more effective when the larynx participates in the affection. Of the numerous lauded destroyers of fungi, of the strongly irritant and caustic remedies which are intended to cut the process short, or to diminish the swelling (nitrate of silver, carbolic acid, flowers of sulphur, etc.), not one has proved of value ; a fact which is attested by the continual introduction of new remedies. (For further information consult the section on Croupous and Diphtheritic Inflammation of the Soft Palate.)

Condition of the Naso-pharyngeal Cavity and the Lower Pharynx in Tuberculosis.

In tuberculosis the mucous membrane of the two cavities is rarely altogether normal. In most instances there are diseased conditions of different sorts. Only a part of them possess a specific character.

The frequency of *anœmia* of the pharyngeal mucous membrane in the commencing stage of tuberculosis (Smith) has already been mentioned. The frequency of *simple* and of *follicular catarrh* of the lower pharynx and the naso-pharyngeal space is very noticeable. In the latter locality they are often associated with swelling, which may attain a high grade.

In special cases I have found *suppuration of the follicles* less frequently on the posterior wall of the lower pharynx than in the naso-pharyngeal cavity, where, indeed, the greater portion of the follicles may undergo destruction. This leads, in the first place, to the formation of small abscesses and ulcers, and subsequently to cicatrization, to shrivelling of the parenchyma, with numerous contractions of the free surface.

There occurs, not altogether unfrequently, in both cavities (the lower pharynx and the naso-pharyngeal cavity), in tuberculous individuals, an affection attended with destruction of tissue, in which there is nothing characteristic, and which also occurs in patients affected with other diseases (especially scrofula, congenital and contracted syphilis), and also in those who are, to all appearances, healthy. This is the tubercularizing, caseous, scrofulous inflammation of the follicles—*pharyngitis scrofulosa.* In the lower pharynx, these follicles are generally attacked in groups, in great numbers; they assume a color varying from yellowish-gray to gray, are of a dry caseous consistence, or are found in the act of breaking up into fatty detritus. Numerous small ulcers are formed by the destruction of the follicles which lie near the surface. The destruction of those which are deeply imbedded leaves numerous roundish spaces behind in the parenchyma. This leads to rapid and oftentimes extensive destruction at the localities affected, which is completed by the absorption of the remnants of tissue between the groups of follicles. Such ulcerations of the lower pharynx, mostly very extensive, and principally upon the posterior wall, have been described by Isambert, Bryk, and others. In the cases observed by these authors, they occurred without manifesting any special symptoms (by confluence of follicular ulcers), extended further, and led, among other results, to adhesions with the soft palate. On account of their destructive tendency, they are brought into association with scrofula, but especially with syphilis, and with a combination of the two. I have seen a similar condition during life under the conditions narrated, in an otherwise healthy girl, who became diseased in this way several times during the course of four years, and in whom, finally, without any special local symptoms, there ensued an adhesion of both palatine arches with the posterior wall.

Rindfleisch describes ulcers which have their origin in this scrofulous inflammation, in hyperplasia and destruction of the pharyngeal follicles, as an ordinary complication of tuberculous ulceration of the larynx.

In this manner ulcerations of considerable extent occur, especially in the pharyngeal tonsil and on the posterior wall. In the former locality they appeared to me sometimes of a caseous consistence, evincing a destruction which is not limited to the follicles, but which affects the cytogenetic layer in general.

I have found *ulcers* in the naso-pharyngeal cavity, principally in tuberculosis, the larger ones exclusively in such cases and in some syphilitic cases. I have rarely seen large ulcers here. In the literature accessible to me, I likewise find very sparse reports concerning cases observed during life. I cannot, therefore, substantiate the view of von Troeltsch (Lehrbuch, third edition), that the by no means rare occurrence of very extensive ulcerative conditions in the upper pharyngeal space is also observed independently of association with syphilis.

The ulcerations which I have observed in tuberculosis, especially in its advanced stages, and simultaneously with intestinal ulceration, generally affect the pharyngeal tonsil and the posterior wall together, or both in immediate connection, or only one portion of them. I have also seen ulceration in the recessus pharyngis and in the tubal projections, which in one case I saw destroyed down to the level of the lateral walls. On the vault of the pharynx the ulceration readily acquires a great extent. The destruction has repeatedly extended almost over the entire tonsil and the contiguous portions of the posterior wall—in one instance at the same time over the recessus and the posterior surface of the tubal projections, which were deeply gnawed in their upper portions. Ulcers occur of two centimetres in diameter ($\frac{4}{5}$ of an inch), antero-posteriorly and transversely. The depth varies (from one to three and even five mm.) (from $\frac{1}{25}$ to $\frac{1}{5}$ of an inch), being unequal at different points, deeper in some places, and decreasing in depth towards the edges, etc. There are flat and kettle-shaped ulcers, and some with very irregular surface. The bottom of the ulcer is raw, mostly covered with fine gray, reddish-gray, yellowish-gray, or yellowish nodules, which may also be observed in the tissues adjoining them. The border of the ulcer is usually sharp, also serrated, and with steep or undermined edges. Sometimes there is a gradual transition throughout the entire circumference or at certain portions. I have seen the bottoms of ulcers and the surrounding parts strewn with small hemorrhages, covered with fatty or caseous detritus, or with a thick adhesive layer, and frequently with greenish-yellow mucus, which hung far down on the posterior wall of the lower pharynx. The manner in which compensation of destruction may take place is shown by the indurated thickening of the tissue in many ulcers, as well as the simultaneous occurrence of cicatrices with such thickening. The individuals affected were mostly between twenty and thirty-five years of age. But even in a child nine months old I found a lentil-shaped loss of substance in the pharyngeal tonsil.

Many of the ulcers examined exhibited nothing characteristic, nothing satisfactory concerning their production. Others presented a follicular origin. In two cases, one of which was the one that showed ulceration on the posterior surface of the tubal prominences, I was enabled to authenticate the specific process, in the edges of the ulcers and in the tissue immediately adjacent, in the formation of fresh miliary tubercles, with one or several giant-cells within the reticulum of their nodules.

In some cases, in which there were extensive ulcers in the pharyngeal tonsil, the middle ear was wholly intact (so also in those associated with commencing destruction of the tubal projection from behind forwards), or diseased on one side only (catarrh—defect in the tympanic membrane in one instance). In the case which had proceeded to ulcerative destruction of one tubal projection, with cicatrized partial destruction of the other, I have found both orifices so closed by swelling as to be reduced to a small linear slit. In consequence of the rarefaction of the air thus produced in the hyperæmic tympanic cavities, which were partially filled with a moderate accumulation of mucus, the tympanic membranes were pressed so far inwards that the handle of the malleus lay upon the promontory.

The presence of tubercles has not yet been authenticated in reference to the middle ear (except the ulcers upon the tubal prominences in one of my own cases) and the tympanic membrane. Still the clinical observation, in some cases of tuberculosis, of rapid and almost symptomless destruction, especially of the tympanic membrane (von Troeltsch, the author, Schwartze), seems to indicate it. On the other hand, chronic inflammation of the middle ear is frequent in such cases and in scrofulous subjects, often exhibiting great resistance to local treatment, usually associated with secretion of pus, and sometimes leading to destruction, even of the osseous portions.

Little is known concerning the occurrence of tubercles in the *lower pharynx.* B. Wagner describes as true tuberculosis of the pharynx some projecting grayish red nodules, from the size of millet-seeds to that of hemp-seeds, and isolated flat ulcers on the upper half of the pharyngeal wall and on its sides, partly round and partly irregular in conformation, produced by confluence and covered with a caseous pus. They were found in connection with numerous ulcers at the root of the tongue and in the soft palate, in cases of tuberculosis of the lungs, liver, spleen, and kidneys. On microscopic examination, he saw small cells and nuclei in these nodules, mostly shrivelled and in course of destruction, and especially finely granular and fatty detritus; and in the pharyngeal mucous membrane a copious development of nuclei, principally about the enlarged mucous glands. The follicles were little or not at all swollen.

E. Wagner observed an analogous process in two cases in which, during life, pulmonary phthisis was diagnosed with secondary syphilis of the lower pharynx. In one case there was considerable thickening of the mucous membrane due to hypertrophy—that is to say, new formation of reticular connective tissue. In the other there was ulceration (large ulcer of the posterior pharyngeal wall), with the formation of partly diffuse and partly follicular tissue, in the larynx and other organs, and especially in the palate and lower pharynx, where at some points it penetrated between the muscles.

According to O. Weber, the ulcers occurring here on the posterior and lateral walls in tuberculosis have the greatest similarity with the corresponding intestinal affection. "They have a caseous, broken-down floor, undermined hyperæmic edges, in which new tubercular deposits are imbedded in various stages of development, and which rapidly disintegrate and cause necrosis of the mucous membrane lying between them." He saw three cases rapidly terminate fatally, in which there was coexisting tuberculosis of the lungs in part, and of the intestine in part. Concerning one case, in which the process had been but little developed in the lung, he is inclined to attribute the main cause of the rapid running down to the large ulcers in the lower pharynx. Under certain circumstances the affection is very painful (in contradistinction to scrofulous ulceration), and associated with difficulty of swallowing.

Concerning the *treatment* of the diseases of the naso-pharyngeal space and the lower pharynx in tuberculous subjects, the principal object is the fundamental disease itself. In addition, the same measures as are employed in chronic catarrh of the cavities come into play, with narcotic and emollient inhalations to relieve the pain of pharyngeal ulcers. Strongly irritating and caustic applications to tuberculous ulcers are rather hurtful than useful, as experience teaches in reference to other mucous membranes. According to Türck, it is impossible to restrain the development of diseases of the larynx dependent upon tuberculosis; and he saw cauterization do visible injury in many cases. Niemeyer, Schnitzler, Siegle, and others express themselves in a similar manner. O. Weber expressly advises the local use of nothing beyond a disinfectant mouth-wash, and to omit the use of gargles and especially cauterizations. Even in scrofulous ulcerations, such procedures as are recommended by Isambert (pencilling with tincture of iodine, with a five-grain solution of chloride of zinc, or with a one- or two-drachm solution of chromic acid), appear to be of little use. I have seen no beneficial results in such cases even from severe cauterization with pure chromic acid and with nitrate of silver.

Condition of the Naso-pharyngeal Cavity and the Lower Pharynx in Syphilis.

A considerable part of the diseases which affect the *naso-pharyngeal space* in syphilis assume the form of *catarrh;* in other instances they go on to destruction of tissue and to adhesions of parts. They have as yet received but little attention. Only isolated observations are at our command.

Some special conditions have been learned with the pharyngeal mirror. Thus, syphilitic *ulcers* on the vault of the pharynx were observed by Türck in two cases. He saw, at the same time, ulcerous formations on the posterior lower pharyngeal wall, which structure, according to him, is much more frequently alone diseased in this manner without extension of the destructive process upon the upper cavity. Semeleder found ulcers in three syphilitic individuals: one very large one on the upper and posterior wall (eventually, also, ulceration and perforation of the soft palate); one, undergoing granulation, located in the region of the tubal orifice (with similar conditions coexisting on the posterior surface of the soft palate, and on the posterior border of the nasal septum); and, in the third, extensive ulcers on both sides of the naso-pharyngeal space (with ulceration on the posterior portion of the pharyngo-palatine arch). In one case observed by the author, there was a tolerably large ulcer in the naso-pharyngeal cavity *alone*, with intense congestion of the cavity and of the lower pharynx, and very copious secretion of mucus. The ulcer, deep, with lardaceous bottom and sharp outline, occupied the anterior portion of the pharyngeal tonsil, and reached down upon the right ala of the vomer. Only the occasional discharge of sanguinolent mucus by the nose and mouth had prompted the patient to seek relief. After repeated inunctions, and rigid cleansing of the parts (nasal douche), recovery took place. A slight depression remained behind, corresponding with a cicatrized superficial defect upon the right process of the vomer. In consequence of this, the right posterior naris appeared larger and vertical, and the septum as though pushed to the left side above. The author has under treatment a large ulcer on the roof of the naso-pharyngeal space.

Concerning the pathologico-anatomical condition of things, the author can report something from his own observations. In three-eighths of the cases in which decided syphilitic diseases existed in other mucous membranes, in the skin, or in the bones, or where the evidences of such diseases were visible, the naso-pharyngeal space was also implicated in one way or another. Thus I found in one case a pointed condylomatous vegetation at one tubal orifice, and in another, a slit-shaped constriction of one orifice from hypertrophic development of the mucous membrane. Ulcerations were seen in few cases, either in the pharyngeal tonsil, or on the lateral walls (recessus, projection, or orifice of the Eustachian tube), but cicatrices in various places were seen more often, several times together with ulcers.

In one tube there was a constriction of the orifice by a flat cicatrix (a case of mucous catarrh of the middle ear). In one case, besides a large radiated cicatrix of the pharyngeal tonsil, there was a similar correspondingly smaller one at the anterior side of the left tubal orifice, a firm cicatricial constriction of the right tube, and a larger and deeper ulcerous defect at the upper half of the right tubal prominence (with marked depression of the tympanic membrane, hyperæmia, and hydrops ex vacuo of the tympanic cavity). In another case, where there were cicatrices on the tonsil and the posterior wall, there was a cicatricial constriction (slit-shaped) of the opening of the tube, on the right side of the naso-pharyngeal space; a large deep cicatrix in front of it, with cord-like projections; a defect of one portion of the prominence, covered with cicatricial tissue, and with it an ulcer upon the prominence, and another just beneath the orifice. The left lateral wall was formed of a thick, dense, whitish, cicatricial layer, which showed strands and perforations here and there, presenting, in general, a uniform surface lying one mm. (one-twenty-fifth of an inch) below the level of the nasal mucous membrane. Here, an irregular slit, two mm. (one-thirteenth of an inch) in height and one mm. (one-twenty-fifth of an inch) in breadth, represented what remained of the tubal orifice. It was still possible (on the preparation, by accident) to drive air into the tympanic cavity, by holding the beak of the catheter over it in a certain position. (There was mucous catarrh of the middle ear, on the right side, with hypertrophy and multiple cyst-formation; muco-purulent catarrh of the left side, with hypertrophy, and with defect in the tympanic membrane.)

Gruber has described a complete cicatricial occlusion of the orifice of one Eustachian tube, after syphilitic ulceration. He saw, in addition to defect of the nose, an adhesion of the soft palate to the posterior wall, and entire destruction of the prominence of the tube, the orifice of which was closed by cicatricial tissue. Such complete obliteration occurs only very exceptionally, even when due to other causes. Cases have been reported, one each by Otto and Lindenbaum (see above), in which the manner of occurrence is not distinctly characterized.

In a case reported by Virchow, "there were such large cicatrices upon the mucous membrane of the pharyngeal and nasal cavity, that the mouth of the Eustachian tube was almost entirely closed." The adhesions of the soft palate, to be spoken of further on, do not, in and of themselves, produce any alteration of the tubal opening, or any disturbance of hearing (case of Wilde), even when they amount to complete separation of the upper cavity from the lower one. In those frequent cases in which hardness of hearing exists in syphilitic individuals, it is due in most instances to the ordinary diseases of the middle ear. They are transplanted from the naso-pharyngeal space, as chronic catarrhs or inflammations; or they occur secondarily, in consequence of occlusion of the tube. Such a condition takes place (apart from accumulation of secretion, swelling or hypertrophy of the mucous membrane), when the mouth of the tube is constricted by the formation of cicatrices, or is drawn together by cicatrices in the vicinity, by cicatricial bridges of tissue, or by immediate adhesive contact with that portion of the palate which is nearest to it. (An impediment to the action of the muscles of the tube may also result from adhesions in some cases.) In consequence of such alterations in the region of the orifice of the tube or in the orifice itself, the catheter may not pass beyond the choanæ at all, or it does not reach the orifice, or its introduction is only accomplished with difficulty and in exceptional cases. By the forcible introduction of air, or by slitting the tympanic membrane, the equalization of the pressure of the air within and without the tympanic cavity may be temporarily established. The latter procedure also permits the escape of the secretions, accumulated for the most part on account of the impediment to their discharge, and thus leads to a remarkable though temporary improvement of the frequently very marked hardness of hearing. A *permanent* favorable result might perhaps be reached in some cases by excision of the greater portion of the tympanic membrane. The excision of a small piece, in order to establish a permanent communication between the tympanic cavity and the external auditory meatus, does not accomplish the result, owing to the well-known great regenerative properties of the membrane, as shown in traumatic defects. Concerning the insertion of a perforated eyelet (Politzer), or of a hollow perforated ring enclosing the handle of the malleus (Voltolini) into the tympanic membrane to render the opening permanently patulous, there are no sufficiently satisfactory observations.

Hardness of hearing, mostly of a marked degree and of sudden commencement, is sometimes dependent upon a syphilitic disease of the labyrinth. Such affections I have seen promptly cured in several instances by suitable general treatment; while the same treatment is, at most, of but limited value, and often of none at all, in simple complications on the part of the middle ear, and has no influence whatever in those conditions of the orifice of the tube dependent upon definitive alterations.

It should be remarked, in this connection, that simple fissure of the uvula cannot give occasion to impeded action of the muscles of the tube; but this may be the case, even to insufficiency of the muscles and all its resultant effects, in deep clefts of the soft palate, or in that form of the deformity also involving the hard palate—in consequence of the modified attachment of the muscles. The crossing of the levator muscles in the uvula (Semeleder) gives an altered configuration to the lower border of the mouths of the Eustachian tubes. Still, differences doubtless occur here—perhaps more or less favorable secondary insertion of the muscles, principally of the here especially important tensor, the special opener or abductor (von Troeltsch) of the tube; for disturbances of hearing do not always occur under such conditions. I have thus seen a student with true cleft palate (wolf's throat) whose hearing was very acute. When other congenital anomalies of the ear itself are present, in addition to such a defect, as a matter of course there will be important disturbances; as in a case of rudimentary development of both tubes, with obliteration of the mouth of one, described by Gruber.

At the commencement of syphilis, and not unfrequently in its later stages also, diseases frequently occur in the *lower pharynx*, which are hardly to be distinguished from ordinary catarrh; there may be pronounced swelling or an entire absence of swelling, and there is redness, often with dryness in the throat, and attended with difficulty of swallowing, if there is infiltration—syphilitic *catarrh*, or erythema. In some cases there are to be found at the same time white patches, due to a *thickening of the epithelium* from proliferation and suppuration, which do not present anything characteristic, and which are to be met with in the same way also in the course of syphilis and in non-syphilitic catarrh. In the same manner there may be small losses of substance—*erosions*—in the epithelium.

The *papules* and *patches* are of more importance. They are roundish, elevated, white spots of the mucous membrane, strongly infiltrated with cells, and may lead to ulcerations. In the same category we include the *nodes*, gummata, circumscribed infiltrations which undergo rapid and extended destruction, giving rise to deep and large *ulcers*. The tones of the voice acquire a nasal clang in cases of defect of the soft palate. Many patients learn to avoid the passage of ingesta upwards into the naso-pharyngeal space.

Repair takes place, in the further course of the disease, by the formation of cicatrices. Some of these are radiated, excavated, and productive of a wrinkled drawing together of the contiguous surfaces. In other cases there is a direct adhesion of parts previously separated, or a connection of these parts by membrane-like cicatricial tissue stretched between them.

The frequent *adhesions* of the soft palate with the pharyngeal walls, which have been comprehensively described and discussed by Ried, present important variations. Either the posterior arch of the palate only (one, or both), or the uvula only (though this is rare), is grown fast to the posterior wall. In such cases there are hardly any symptomatic disturbances (only slight modification of speech). The adhesion may affect the soft palate also (oftener on its flat surface than at its border alone), in such manner as to leave but a very limited communication between the upper and lower cavities. The opening is usually found at the site of the destroyed uvula, or behind it, when it has been retained, as is but seldom the case. In these instances a portion of the food may pass into the nose, though some patients learn how to prevent this ; but breathing through the nose is rendered more difficult, and speech is altered. The adhesion may be complete on all sides. Nasal respiration is then wholly abolished; smell and taste are impaired; the mouth and lower pharynx are dry; and the nasal secretions cannot be removed in the usual manner.

Such adhesions occur also in other pharyngeal affections which are associated with injury to the epithelium, or with ulceration—in lupus, scrofulous inflammation, diphtheria, and phlegmon. The formation of cicatricial membranes is very infrequent in these affections or in syphilis; they may produce stenosis and partial occlusion of one portion. In a case observed in the clinic of Langenbeck, the entrance to the larynx was so far closed that only an aperture the size of a pea remained, the obstruction being a membrane reaching from the posterior wall of the lower pharynx to the root of the tongue, the closure of which occasioned dyspnœa (Trendelenburg).

The syphilitic affections of the cavities, even the most important ones, often progress without prominent *symptoms*, and without essential annoyance to the patient, so that the commencement of destructive processes is often overlooked. Sometimes there are vague, painful sensations, occasionally severe pain (in ulceration), increased by the movements of deglutition, and sometimes referred to the ear (in ulceration of the tubal region, but also in ulcerations of the deeper portions of the lateral wall). In some cases of ulceration in the lower pharynx, the annoyances are limited to an unpleasant sensation in taking dry food (as bread). In syphilitic affections of the naso-pharyngeal space, headache, or an uneasy sensation in the head is sometimes felt; and hardness of hearing and subjective aural sensations are frequently complained of. Not unfrequently there is a foul odor of the air expired through both cavities in cases of ulceration and in cases of want of cleanliness.

Diagnosis.—Direct inspection of the lower pharynx, and examination of the naso-pharyngeal space by means of the mirror, reveal the existence of alterations in general. The latter method of examination, in many instances, affords opportunity for timely detection of processes which might otherwise terminate undetected in the destruction of important parts (soft palate, nasal septum, etc.). The individual alterations are not in themselves sufficiently characteristic to be classified as local manifestations of syphilis without further investigation. Greater certainty is secured by considering the manner of their appearance, their further transformation, the condition of the immediately adjacent parts (want of a proportionate participation in those parts, such as exists in other diseases), the condition of the lymphatic glands, as well as the presence of corresponding affections of other mucous membranes and of the skin, and, finally, the previous history of the case.

General *treatment* is not to be dispensed with in most cases. In cases of ulcerous destruction, the inunction treatment and the administration of calomel are justified. The avoidance of local irritation (smoking, snuffing, certain articles of food) is a prominent essential towards improvement and cure, and so is a constant, careful maintenance of cleanliness of the parts (by nasal douches, gargles, and the pharyngeal bath). Both these points are also of well-known importance in a prophylactic point of view. Astringent fluids are advantageously used for cleansing purposes. Von Sigmund recommends, for use by the nasal douche, chlorate of potash, from one to two and a half drachms; permanganate of potash from twenty to forty grains, or carbolic acid from twenty to sixty grains, to eleven and a half ounces of water. For the lower pharynx, he recommends alum, zinc, and tannin; and in ulceration of the lower pharynx the same solutions as used in the nasal douche, only proportionably stronger. He finds corrosive sublimate, one grain in six ounces of water, most efficient for the pharynx, and the same quantity in eleven and a half ounces of water for the nasal douche (to be used only by very careful patients). In cases of gummous softening he uses iodide of potassium, from one to two drachms to eleven and a half ounces of water, and diluted tincture of iodine in the same proportion. He also attributes great value to the frequent daily application of sprays of alum, zinc, and tannin.

The results of the operative treatment of adhesions already established are but little successful, according to Ried. To prevent their occurrence, it is recommended, in all affections of the lower pharynx associated with loss of epithelium or with ulceration, to employ cauterization with the nitrate of silver, and to insert strips of linen, or wads of charpie, saturated with some astringent solution.

In some cases of more extensive defects of the palate, the obturators scientifically constructed by Süersen are of use, as they replace, as it were, the missing valve of the palate. They do not quite touch the posterior wall of the lower pharynx, and therefore they admit of the production of nasal sounds. The closure of the remnant in swallowing, and in speaking the remaining sounds, is effected by the horizontal, cushion-like projection at the corresponding portion of the posterior wall (by the action of the upper constrictor—Passavant), which is formed by these efforts.

Hyperplastic Catarrh of the Naso-Pharyngeal Cavity and the Lower Pharynx (Hypertrophy).

In the course of chronic disturbances of nutrition of the mucous membrane of both cavities, there are frequently hyperplastic processes which here present some peculiarities. They are characterized by augmentation of the normal tissue elements, by increase of volume and alterations in the conformation of the affected parts.

In the naso-pharyngeal space it is especially the cytogenetic layer which, in its hypertrophic development, diminishes the calibre of the cavity, and gives occasion to disturbances of various important functions. The increase of the mass is here either about equal in degree in all parts, or it is especially prominent at several points, most frequently in the pharyngeal tonsil. It is mostly of such a nature that the projecting portions have merely a moderately enlarged appearance, a thickening of their covering or of their substance (tonsil) in various grades—the deepened portions undergoing contraction or flattening. Sometimes, however, the increase is so extensive that there is a hanging down of the tonsil over the upper portion of the choanæ, or also (though seldom) laterally over the tubal outlets, with a constriction of these orifices and of the recessus, or even an entire obliteration or growing together of the sides of the recess. The free surface is thereby altered in configuration in various ways. In many cases the original form (flat, or flatly semi-globular), and the arrangement (in longitudinal divisions), are tolerably well maintained. It is laid in longitudinal ridges, or combs, of various heights, which, stretched or twisted, increasing and decreasing also in height and breadth, course antero-posteriorly or obliquely, and are separated by fissures or lacunæ of varying depth. In other cases the hypertrophied longitudinal ridges are grown together here and there or in many places. Then there is a coarse network, or a tolerably smooth surface, interrupted by many small slit-like or roundish openings—the entrances into the remains of the lacunæ. The tonsil appears, under the conditions thus far described, of an almond shape in general, semi-globular, or in the form of a flat extension of nearly uniform thickness. In some cases the difference in the hypertrophic development of the ridges, especially in breadth and height, is so marked at special points, that it occasions the formation of polypoid protuberances, tongue-like or club-like, in part with excessive thickening of their free portion, and with a lobulated appearance of the surface. But the elegant papillary structure exhibited in many polypi of other mucous membranes, is never found in these projections. This modification of polypoid hypertrophy, due to the local conditions of the tissue (cytogenetic tissue), either affects the tonsil exclusively, or is here most strongly pronounced.

The hypertrophic covering of the lateral walls presents for the most part a uniform surface, uninterrupted except by prominence of follicles. In individual cases the cytogenetically clothed places are raised in small, roundish, flat, and even semi-globular or tongue-like protuberances arranged in strips. At the parts which are not cytogenetic—the anterior tubal regions—the increased thickness of the mucous membrane is sometimes associated with the formation of folds. The roundish prominences are found on the tubal projections and in the recessus; while the ridges, usually of slight development, are more frequently found in the latter, very seldom in the tubal prominences, running here obliquely or horizontally, but only in a rudimentary manner. The largest that I ever saw were small combs, two and one-half mm. (one-tenth of an inch) high and six mm. (a quarter of an inch) long, which in one case stretched from below upwards to both tubal orifices, while, in another case, there was a similar comb at the same point, but only unilateral with partly round and partly tongue-like or globular projections on the tubal prominence. In some instances there were rafters or solid high bridges of tissue which united the exterior border of the pharyngeal tonsil with the tubal protuberance. A pectinated elongation of the latter structure, such as may take place in acute swelling, I have never encountered in hypertrophic conditions.

There is here sometimes a uniform increase in volume with retention of the original form, or a polypoid projection, an hypertrophic process associated with the formation of polyp-like elevations. The method of development of these formations, their configuration, is influenced by the cytogenetic consistence of the mother-tissue which is unfavorable to the production of mucous polypi and papillomata.

Besides the alterations of level described, which may be very varied, the hypertrophic mucous membrane of the cavity shows, in most instances, still further peculiarities of the surface. The surface is often perforated in a sieve-like or sponge-like manner, strewn with holes and fissures—the mouths of dilated and enlarged mucous glands, the openings of the follicle-like depressions which may here be formed, not only in the tonsil and in the recessus, but also on the tubal prominences. Projections of the surface from enlarged follicles in the cytogenetic portions, by cystoid enlarged follicles and by dilated acinous glands in all parts, are very frequent. True cysts are more infrequent. Very often there is a very dense coating of mucus.

Within the hypertrophic cytogenetic layer there is also, in most instances, an increase of the ordinary connective tissue which accompanies the glands and vessels. The parenchyma acquires a greater consistency by the production of numerous firm connective-tissue bundles, but never, except in extended absorption of the cytogenetic tissue and in cicatrization, a density such as the hypertrophic tonsils of the palate undergo under certain circumstances. A large hard pharyngeal tonsil does not appear ever to be produced.

It is still necessary to compare the descriptions and views of other observers with the representation given above of the different forms—among others, polypoid forms also—in hypertrophy of the cytogenetic expanse in the naso-pharyngeal cavity. My own descriptions are based chiefly on anatomical examinations, but also on exploration with the mirror and the finger. There cannot be any doubt, from their description, that Czermak's "cock's comb protuberances" at the mouths of the tubes, that the vegetations so vaguely described by Türck, and that the two cases of "polypi" from the roof, seen by Semeleder, all belong in this category (Meyer). Voltolini was the first (in 1865), by means of the mirror, to observe comb-like, club-shaped, or berry-shaped formations at points on which the cytogenetic tissue is found to be diffusely distributed.

He justly recognized this as an hypertrophic process, and he called the elevations "polypoid vegetations of the mucous membrane." He was also the first to undertake their removal by operation (by galvano-cautery). Löwenberg has reported five cases (1867), which he classed under the head of "pharyngitis granulosa." Three of these are to be included here (teat-like tumors, in one instance, with flattish hypertrophy of the pharyngeal tonsil). Wilhelm Meyer has devoted very careful study to these conditions. His views are founded in part on palpation of the cavity, and in part upon the evidences furnished by the rhinoscopic mirror, but not upon conditions found in the corpse. He met principally comb-like or teat-like formations. The comb-like formations appear chiefly upon the vault and the upper portion of the posterior wall (corresponding, therefore, to the pharyngeal tonsil), and are described as ordinarily numbering from four to eight, parallel to each other (thus altogether corresponding to the normal type of the ridges). The fimbriated variety are frequently found on the vault, and also on the lateral walls, to which they lend "a coarse glandular" appearance. Meyer also considers even the configuration of the combs and fimbriations of the tonsil as preformed in the original lay of the tissue, the fimbriations of the lateral walls being produced from the histological elements of the cytogenetic tissue. He does not give sufficient prominence to the existence of somewhat large, and simply swollen, pharyngeal tonsils. He designates both forms, combs and fimbriations, under the common appellation of adenoid vegetations, growths of adenoid cytogenetic tissue, which is in no case a fitting designation for hypertrophied and swollen longitudinal ridges, but might better suit the more developed fimbriæ.

The frequency with which Meyer found these formations among his countrymen, the Danes (one hundred and seventy-five cases in five years, one hundred and thirty of them with aural affections), is remarkable. He includes, as already mentioned, all enlarged tonsils, and does not exclude, in a definite manner, those affected only with swelling. Perhaps unfavorable climatic conditions come into play as the cause of this national peculiarity; for retro-nasal catarrh, and the catarrh of the middle ear dependent upon it, are especially indigenous on the northern coast.

Voltolini declares that the occurrence of these hypertrophic formations in Germany "is not at all as frequent as appears to be the case in Denmark." He reports four cases operated upon by himself. The author has frequently seen temporary increase of volume of the tonsil (swelling), not unfrequently a permanent increase (hypertrophy) to a slight degree, occasionally a moderate degree of increase (so that it overhangs the upper third of the vomer), and only in very rare cases an increase in the highest degree (covering half the choanæ and more). The author has had occasion to operate only twice in the course of nearly ten years.

In the lower pharynx, the hypertrophied mucous membrane appears thickened in various degrees, often protruding in welts or folds. A partial welt-like hypertrophy of the posterior wall in the laryngeal region, associated with dyspnœa, has been observed by Semeleder and by Lewin. The vessels are dilated, and the acinous glands are dilated and enlarged, but seldom transformed into cysts. The connective tissue elements are increased, and especially the follicles, which are here for the most part large, and exist in great numbers—pharyngitis follicularis, seu granulosa hypertrophica. In some cases there is also a new formation of tubular glands (from pathological formation of folds), especially in the upper portion of the posterior wall. The wall may in this way acquire a spongy appearance, such as likewise occurs in the pharyngeal tonsil. In consequence of this and of the fact that the normal inequality of thickness is done away with by the hypertrophy of the contiguous portion of the posterior wall, the line of demarcation between these two structures may become obliterated.

On the lower pharyngeal walls, especially the posterior wall, there is produced, in certain cases, an increased thickness of the squamous epithelium, which is associated with a grayish-white coloration, with or without elevation from newly-formed papillæ. The secretion is often increased.

The significance of the hypertrophy resides chiefly in the fact that it keeps up the chronic catarrh from which it mostly takes its origin, and renders its cure or improvement more difficult. Furthermore, interference with important functions may be produced by hypertrophy of certain parts. The manner of the production of such disturbances has already been discussed several times, particularly in the description of catarrhal swelling. It is principally the increase in volume of the mucous membrane, associated with constriction or with occlusion (towards nose and ear) of the cavity, which comes into consideration here.

The *symptoms* by which hypertrophic conditions of the lower pharynx are announced are in general those of chronic catarrh, and have been discussed in that connection. The same holds good with regard to the naso-pharyngeal cavity. The hypertrophic development of the part is attended by the same manifestations as those of the corresponding degree of simple parenchymatous swelling in catarrh. Complete abolition of respiration through the nose seldom occurs in these cases; alterations of speech are somewhat more frequent. Symptoms referable to the ear are more frequent, either in consequence of direct extension of disease, or, secondarily, from occlusion of the tubal orifices. This occlusion may be due to constriction from increase in its own volume, or to its obstruction by hypertrophied contiguous parts.

With reference to the palatine tonsil, no matter what size it may have acquired, the anterior lip of the tube cannot become pressed against the posterior lip, either by direct pressure, or by the pushing upward of the posterior palatine arch or the contiguous mucous membrane. It does not even take place from hypertrophic thickening of the velum of the palate (von Troeltsch). The anterior lip is firmly adherent to the pterygoid process. If constriction occurs under the conditions mentioned, it must take place from below. A nearing of the anterior to the posterior lip occurs only by participation of the mucous envelope of the former in swelling and hypertrophy. Under these circumstances there rarely results a complete obliteration of the well-known wide orifice, and then it occurs more in the depth of the funnel; and an actual obstruction by the hypertrophic tonsil which overhangs it only occurs when the lateral combs are also developed to a great degree. On the other hand, occlusion of the middle ear occurs readily and frequently, either temporarily or for a longer time, from the easily retained and accumulated secretions, even in less pronounced diminution of the calibre of the cavity and of the tubal orifice.

The polypi met with not unfrequently, and often simultaneously with hypertrophic conditions of the nasal mucous membrane, take their origin in great part from the posterior extremity of the lower turbinated bone. After considerable development, they may (if of pretty firm consistence) reach the orifice of the Eustachian tubes and even occlude them, and perhaps actually close them by reason of simultaneous congestive conditions of the parts. Or they may hang down pouch-like beyond the veil of the palate (if of gelatinous consistence). Then, even when they are of considerable size, the tubal orifices may remain free and the hearing intact, as I have verified in a case examined with the mirror, in which there were such polypi upon both sides.

Diagnosis.—A uniform increase of thickness of the mucous membrane of the lower pharynx is not readily recognizable, but the presence of projections or of groups of enlarged follicles is more readily recognized. Only the higher grades of hypertrophy of the lining membrane of the naso-pharyngeal cavity announce themselves by distinct clinical manifestations (especially by abolished nasal respiration). The same manifestations, however, occur also in other diseases of the nose associated with impermeability, as well as in cases of larger morbid growths in the nose and in the naso-pharyngeal space; and also *after* such diseases, as the result of habit, even when the nose is again permeable. A direct examination of the parts, therefore, is hardly ever to be dispensed with. The examination by touch with the finger may afford evidence concerning the location, form, and size of decided prominences, and, within certain limits, also concerning their points of origin. Rhinoscopy, on the other hand, reveals, in addition, their color, the presence and condition of secretions, and, in a more certain manner, the configuration of the surface. Great care is necessary in both methods of examination in interpreting what is seen or what is felt, and especially in estimating relations of size.

According to Meyer, the presence of hypertrophic formations often presents insuperable obstacles to the employment of rhinoscopy. Voltolini, on the other hand, advocates examination with the mirror in just these cases. I myself have also employed the mirror with advantage in my own cases. I have sometimes found the copious retention of mucus an obstacle which temporarily impeded the examination, and which rendered the appreciation of inequalities of surface more difficult. If the mass hangs down lower, it can readily be seen, and, if the position of the uvula is favorable, as it sometimes is, the free portions of the posterior nares can also be seen. In cases of slighter development, of semi-globular form, the author has seen the orifice of the tube and its projecting portion free, the latter strewn with small, flattish prominences. Here there were no prominent symptoms present, even when the upper third of the vomer was overlaid.

The *treatment* is to be directed, on the one hand, towards the fundamental chronic catarrh, and, on the other hand, the object should be to secure, if possible, a retrogressive process in the hypertrophied parts, or to remove or destroy them.

For the lower pharynx the principal indications are to produce a strongly irritant effect upon the parts themselves—

cauterization of the projecting mucous membrane or the hypertrophic follicles with nitrate of silver in substance, painting them with the stronger solutions of the same, or with pure tincture of iodine, or with strong solutions of chromic acid. As a matter of course, this must be done with great care, and always after previous removal of the secretions by brushing, swabbing, gargling, and so on. K. Michel recommends very superficial cauterization of the follicular masses with the galvano-cautery, repeated three or four times. Lewin recommends scarification.

For the naso-pharyngeal cavity the treatment for chronic catarrh suffices in many cases to moderate the annoyances or to relieve them. Even a diminution of the volume of the parts may be effected in that way. Besides simply relieving conditions of swelling, which may also be present in hypertrophic parts, as well as in their neighborhood, a retrogression of the hyperplastic formations may be attained in various degrees. This is favored by the constitution of the tissue elements here concerned. The cytogenetic tissue, which here preponderates over the other new-formed elements, is also disposed to retrogressive processes of various kinds. Thus Meyer, after long-continued use of the nasal douche, saw shrinking, not only of superficial products of moderate volume, but also of the tissue on which they were produced.

Cases occur (very unfrequently; see above) in which considerable increase in bulk of the cytogenetic layer is associated with constriction, or with impermeability of the cavity itself, of the choanæ or of the tubes, and in which the manifestations dependent upon this condition (speaking without using the nose, respiration through the mouth, important and otherwise incurable aural affections) call loudly for relief; cases in which the idea of simple swelling is to be excluded from the diagnosis, and in which careful treatment of the retro-nasal catarrh remains without satisfactory result. Here destructive action and operative procedures are necessary.

For the removal of small and soft masses Meyer recommends frequent cauterization with the pure or mitigated stick of nitrate of silver, kept up for a period of from one to two months. He employs a silver rod, bent so that it may be readily brought in contact with the affected part, and which is introduced from the lower pharynx upwards. The author employs simply a silver probe, which can be bent in whatever manner may be desired. Such an instrument is also recommended by Voltolini in his latest article, in which he states that he has also found cauterization, under the conditions mentioned, effectual. To detach or, rather, crush off larger and more numerous formations, Meyer employs a long-handled ring with a blunt, non-cutting edge, which is to be introduced through the nose and directed against the individual prominences by the aid of the forefinger of the other hand, introduced within the mouth. The operation, which, according to Meyer, not unfrequently requires repetition, is not only disagreeable to the patient, as most manipulations implicating the naso-pharyngeal space are in general, but is also very painful. Reactive swelling follows (Meyer) ; also suppurative inflammation of the cavity and (cases of Meyer and of the author) of the middle ear, with perforation of the membrana tympani.

According to the reports of Voltolini, Michel, and even Meyer himself, the use of the galvano-cautery appears to be much less painful to the patient and more promptly beneficial in its effects. Strong reaction is said not to follow it. The cautery or loop may be introduced through the nose or mouth, guided, when possible, with the aid of the pharyngeal mirror.

Rarefying Dry Catarrh of the Naso-pharyngeal Cavity and the Lower Pharynx (Atrophy).

The lining of these cavities, whether previously normal or the seat of chronic disease, often falls into a condition indicative of diminished nutrition. This occurs most frequently in advanced age, though occasionally in young subjects, often in decrepid individuals or those reduced by debilitating diseases, but often, too, in perfectly well persons.

The mucous membrane is considerably thinner, paler, and, in most instances, dry. Sometimes a few or several dilated tortuous and also varicose veins are found coursing in the otherwise anæmic membrane, especially in the posterior wall of the pharynx, and about the circumferences of the orifices of the Eustachian tubes and of the choanæ. They are dilated by the shrinking or rarefaction of the surrounding tissue, and are prominent in consequence of the thinness of the tissue covering them.

This alteration affects either both cavities in their entire extent, and uniformly or in varying grades, or it affects only one section or one part of a section. In some cases the soft palate exhibits a similar condition, the uvula, "needle-shaped" and tapering to a point, is quite long and hangs down in a relaxed state (von Troeltsch). Very often a pronounced atrophy of the nasal mucous membrane exists at the same time, especially indicated by pallor, dryness in general, and a characteristic stunted, slender and bare appearance of the turbinated bones and the vomer. In the naso-pharyngeal cavity, and in the lower pharynx, the atrophic membrane is constituted in different ways, according to the parts affected.

At the roof of the pharynx and on the sides rarefaction of the cytogenetic tissue is not infrequent. This tissue, which otherwise replaces in good measure the stroma of the mucous membrane, is often, to a considerable degree, lost. A perceptible reduction and with it a decrease of volume, especially in the pharyngeal tonsil, is the rule in old people. Even in younger people (down to twenty-five years of age) I have encountered atrophy at this place, not altogether unfrequently, in most instances associated with diseases which impair nutrition (tuberculosis, carcinoma, and also syphilis). In children I have only rarely seen a small, hardly perceptible pharyngeal tonsil of rudimentary development. As there was always a high grade of marasmus present, in consequence of syphilis or caries, the idea may be entertained that even here there was an atrophic process going on.

Under such conditions the thickness of the pharyngeal tonsil is often reduced to two or to one mm. (one-twelfth to one-twenty-fifth of an inch), as in the new-born babe, or even somewhat less. The cytogenetic tissue is either still present in diffuse and thinner expansion, with the retention of single and occasionally somewhat more numerous follicles, or it remains merely at some circumscribed points, replaced in the main by ordinary fibrillated connective tissue of various arrangements, or the latter tissue exists alone. In this manner the remnant of the pharyngeal tonsil acquires a tolerable degree of density, which may closely resemble cicatricial tissue. The atrophic pharyngeal tonsil is the very place where isolated mucous or colloid cysts are often found, which originate from the follicles or glands constricted in the absorption or retraction of the tissue.

The surface is then either smooth, the folds being obliterated, or it still exhibits, in elevations of minimum height and in shallow furrows, the original arrangement in horizontal ridges and slits, or it exhibits isolated large or numerous small roundish perforations which lead into the remains of lacunæ. If the mucous membrane of the nose is normally thick, or pathologically thickened, with great shrinking of the tonsil, there is, instead of the previous prominence, a flattening to the same level or a depression beneath it, with a sharp line of demarcation at the point of transition. In case of simultaneous atrophy of the nasal mucous membrane, a sharp projecting ledge is formed at the upper border of the choanæ.

On the lateral walls of the naso-pharyngeal space the shrinking, especially of the cytogenetic tissue, produces characteristic alterations. The orifice of the tube gapes widely and is much deepened ; the tubal prominence projects much further forward, and appears as though bare beneath its thinned covering. The fossa of Rosenmueller may undergo marked deepening. In some cases it is traversed by thin connective-tissue strands, like the remnants of a former, much interrupted, spongy enveloping tissue, or a (cytogenetic) tissue connecting its walls in the form of a bridge-work.

The atrophy extends either over the lining structure of the entire naso-pharyngeal cavity, or it is limited to the tonsil or to the lateral walls. In this way the tubal projections are sometimes laid bare while a normal condition of the pharyngeal tonsil still exists, and, exceptionally, even in hypertrophy of the tonsil. On the other hand, again, I have seen follicular catarrh of the posterior wall, the projections, and the recessus, in cases of shrinkage of the tonsil.

On the posterior wall of the lower pharynx the atrophic mucous membrane appears of a pale reddish or pale yellowish color, generally shining, as though varnished, smooth, tense and dry—*pharyngitis sicca.* In the severer grades of rarefaction the color is less pale, because the redness of the muscular bundles of the constrictors shines through. These are seen, often with great distinctness, gleaming through the thinned mucous membrane—in some cases even looking as if they lay exposed, deprived of their covering. In one such case, in the dead subject, I saw numerous white points on the posterior wall, which proved to be encapsulated trichinæ in the muscles, shining through. The mucous membrane was only one-one-hundredth of an inch in thickness. The same degree of tenuity of membrane was also found in other cases examined. The epithelium was normal, the connective tissue long-drawn out, or arranged in waves. Racemose glands were sparse, and follicles were not present at all, or only quite isolated. In one case only, despite otherwise marked shrinking (shining through of the constrictors), numerous follicular prominences were still present. There were also slit-like depressions of the mucous membrane representing the remnants of vanished follicles.

Symptoms.—In many cases, atrophy of the mucous membrane of the lower pharynx occurs without annoyance to the patient; and it often remains symptomless, also, in the upper cavity. Other patients complain of annoying and even tormenting sensations. In the naso-pharyngeal space there is mostly an ill-defined, uncomfortable sensation, which is referred to "the head," or a sensation of dryness which is usually referred to the nostrils (both in the normal and in a pathological condition of their mucous membrane). In the lower pharynx there is chiefly this feeling of dryness which calls forth a frequent desire for moisture, and a tension which renders movements of swallowing uncomfortable and even painful. In some individuals there are burning or stinging pains. These manifestations appear permanently, in the same or in a changeable manner, and in variable severity. They are sometimes rendered worse by many external influences (heat, among other things). Smoking, drinking, and the use of hot and irritating food are not unfrequently well borne, and sometimes thus exert a temporary feeling of improvement, apparently by increasing the flow of blood and inciting secretion.

The *diagnosis* is almost always easy upon inspection of the parts. In the posterior walls of the lower pharynx, which are open to direct view, the pale color, the dryness, the smoothness, the peculiar varnish-like lustre, and the translucent and apparent bareness of the constrictor muscles, are characteristic. In the naso-pharyngeal cavity, digital exploration may convey information concerning the wide gaping of the tubal orifices, the precipitous projection of the tubal prominences, and the sinking inwards of the atrophic tonsil; and rhinoscopy reveals still more concerning the color and dryness of the parts. In one case, with the mirror, I saw the shrinkage appearing on the roof of the pharynx alone; in several individuals (elderly people), there were alterations also on the lateral walls, the ring of veins about the tubal orifices and the choanæ was present, and there was also the above-described similar atrophic condition of the nasal mucous membrane.

The *treatment* is limited to procedures which moderate the symptoms. In the naso-pharyngeal space the nasal douche is useful, with warm, diluted milk, or solutions of salt or muriate of ammonia. The frequent snuffing up of such solutions, also, as well as the use of tobacco snuff, affords temporary alleviation. For the lower pharynx we may resort to the frequent swallowing of small quantities of fluid, the choice of which (among other things, whether hot, containing fruit juice, carbonic acid, etc.) is best left to the patient himself. They should be swallowed after as long retention as possible, with the head bent backwards. Frequent drinking in general, the chewing and gradual swallowing of fruit bonbons, sticks of inspissated liquorice juice, and the like, are also useful. In some cases, annoying symptoms are alleviated for some time by severe irritant influences upon the mucous membrane (pencilling with concentrated solutions of nitrate of silver, with pure or diluted tincture of iodine).

Morbid Growths.

In the *naso-pharyngeal cavity*, in addition to the previously-described polypoid formations due to hypertrophy of the cytogenetic layer (Meyer's adenoid vegetations), we find mucous polypi, fibroids, sarcomas also, enchondromas, and occasionally carcinomas, the *naso-pharyngeal polypi* of the surgeons. They often spring from the roof of the pharynx and also from its posterior and lateral walls, or they grow from the nose or from the upper jaw into the interior of the cavity. The larger growths sometimes have several (secondary) points of insertion.

Symptoms.—Tumors of large circumference produce abolition of nasal respiration and alteration of speech. An impediment to swallowing takes place when they exercise pressure upon the soft palate. This may be crowded far forwards and downwards. From this cause there is a want of closure above in the act of deglutition; in consequence of which, the ingesta, fluids especially, get into the naso-pharyngeal space and into the nose. When these tumors acquire greater extent, the symptoms produced by pharyngeal tumors (increased dysphagia, and dyspnœa) become superadded.

Diagnosis and treatment.—The presence of the tumor, and, when not too large, its insertion also, may be determined by examination with the mirror, the finger, and the probe. The measures employed to remove or reduce such tumors are of the most variable kind (evulsion or twisting off of the smaller ones by forceps or the fingers, ligature, crushing, cauterizations also, undertaken with the hot-iron and the gas-flame, excision with the knife, galvanic cautery, and electrolysis). In many cases, a preliminary operation is requisite (division or detachment of the soft palate, partial or total resection of the upper jaw). Fatal terminations and recurrences are reported, as well as favorable results.

In the *lower pharynx*, the morbid growths take their origin from its walls, or from parts outside of these walls, lying behind them—from the periosteum of the vertebræ, the sheath of connective tissue spread out in front of the vertebræ, and from the lymphatic glands: *pharyngeal and retro-pharyngeal tumors* (W. Busch).

But few observations exist concerning smaller formations. Von Luschka found a *papilloma* measuring three by seven mm. (three-twenty-fifths to seven-twenty-fifths of an inch), which sprang from the posterior surface of the larynx, and hung down into the lower portion of the lower pharynx. Sommerbrodt saw one upon the posterior pharyngeal wall barely the size of a bean; and B. Wagner saw one the size of a hemp-seed in the same locality. In two cases I observed a condition of the mucous membrane of the posterior wall, which might be termed *pharyngitis villosa.* Numerous minute papillomata, from $\frac{1}{250}$ to $\frac{1}{125}$ of an inch in height, gave it a velvety appearance. Fibroids, also, of very small volume, have been observed. Thus Voltolini reports one, the size of half a pigeon's egg, located on the posterior wall. Mosler describes leukæmic tumors of the swollen and congested pharyngeal mucous membrane and the glands belonging to it (numerous pulpy, glistening tumors) under the name *pharyngitis leucœmica.*

The large morbid growths of the lower pharynx are mainly fibroids or sarcomas, and also carcinomas (epithelial cancer and encephaloid) and enchondromas. They may attain a very considerable size, and extend from the cricoid cartilage to the base of the skull, as in a case reported by Fischer (of Innsbruck).

The character of the symptoms occasioned by these tumors depends chiefly upon their seat and their size. Small growths may remain wholly unnoticed, or, from lying in the vicinity of the larynx or in contact with it, may produce cough only, as in the cases reported by Voltolini and Sommerbrodt. In the case of voluminous tumors, the movements of the head generally are impeded, especially in turning it; and there is difficulty in swallowing and in breathing. Even inanition may result or death by suffocation (among others, in a case described by Holt). The cancers are indicated by rapid destruction of tissue, disposition to bleed, suppuration, and recurrence—by swelling of the lymphatic glands, in part by rapid growths, and often by lancinating pains.

Diagnosis.—The presence and consistence of the morbid growth may be ascertained by direct inspection or by inspection with the mirror when it is deeply or laterally situated, and by palpation with the finger and the probe. The larger growths appear covered with a slightly movable mucous membrane, showing numerous dilated veins. This mucous membrane may become ulcerated even in cases of benign tumors of great size (by the mechanical influence of the ingesta which are forced through what remains of the opening of the throat).

Treatment.—Removal with the knife or scissors, evulsion with blunt instruments, and the use of the galvano-cautery, are all rendered very difficult in cases where the growth is of considerable extent. Then there is the proximity to the large vessels of the neck, and the frequent occurrence of severe inflammatory reaction. In some cases which are not adapted to operations (extensive cancerous formations), nutrition by means of the stomach tube, and the operation of tracheotomy may become necessary, as well as the frequent removal or scooping out of rapidly growing masses.

Neuroses.

In reference to the neuroses, the reader is referred to Vols. XI. and XII. of this cyclopædia, as well as to the description of the nervous affections of the soft palate, which have much in common with those of the lower pharynx, as regards their mode of origin, condition, and symptoms. In this place certain neuroses only, of local interest, will be considered.

Anæsthesia of the mucous membrane does not often come under observation, whether existing alone or associated with diminished reflex action.

Hyperæsthesia is more frequent, and is associated with increased reflex irritability (movements of deglutition, gagging, vomiting, cough, and increased secretion). It is as frequent in completely healthy individuals, as in those with local affections. Examination with the mirror, palpation, even an extended inspection of the posterior wall of the lower pharynx, digital exploration of the naso-pharyngeal space, and the introduction of the catheter into the Eustachian tube may be thereby rendered difficult and even impossible. The general (bromide of potassium) or local anæsthetics (chloroform mixed with a solution of morphia in acetic acid, hydrochloric ether, and bromide of potassium), employed for the purpose of diminishing this disturbing sensitiveness, have not proved reliable. Von Bruns has seen advantage from the use of tannin (spraying with a solution of from three to eight grains to the ounce of water, pencilling with a consistent mixture of glycerine and a little water). The psychical influence exerted upon the patient is of value (among other things, diverting the attention, in which category I include Türck's recommendation of continuous gasping respiration, as though "out of breath"); blunting the sensibility of the parts by the patient's practising on himself and gradually accustoming these parts to the necessary manipulations, is also important. The advice of von Bruns, not to undertake these manipulations on a full stomach, is likewise worthy of mention. Finally, the different sensitiveness of different localities, even under normal conditions, is to be regarded in the introduction of instruments, and in mechanical influences exerted at these points. It is greatest on the arches of the palate, upon the posterior upper surface of the epiglottis (von Bruns), and at the base of the tongue; and slightest upon the posterior pharyngeal wall; greater on the posterior surface of the soft palate than on its anterior surface.

The isolated neuroses of the muscles of the palate and the Eustachian tube are little known to those who are not aural surgeons. Some persons can produce a loud cracking noise in the ears, which is similar to the sudden stretching of the tympani membrane caused by the contraction of the tensor tympanic muscle (Joh. Müller), but which occurs in consequence of a rapid abduction of the lateral wall of the tube from the median wall, by the forcible contraction of the tensor palati (Politzer, von Luschka, Löwenberg). Such a cracking, though weak, often occurs in an ordinary act of deglutition.

In other persons the noise occurs involuntarily, from a *clonic spasm of the tensor palati*, sometimes very frequently, and also rhythmically, periodically, very loud, and even audible throughout an entire room (Schwartze), disturbing rest at night (Boeck). The movement of the soft palate accompanying this has been observed rhinoscopically (Boeck). In one case the larynx was raised at the same time (Boeck); in another (Küpper), in addition to this, there were also convulsive movements of the muscles of the eyes, nose, and mouth. In two cases recovery followed electrization of the palatine muscles.

Rüdinger reports a clonic spasm of his own tubal muscles (gaping of the tube) of short duration. Whatever he spoke during its continuance was heard in the affected ear with annoying intensity.

Disturbances of hearing, which appear some time after diphtheritis, may be referred to *paralysis of the muscles of the tube*, if a corresponding condition of the soft palate can be demonstrated, and there are no other important alterations in the ear except diminished atmospheric pressure in the cavity of the tympanum. Inability to open the tube, in consequence of paralysis of the tensor palati, cannot fail to produce this effect, and thus at once produces a purely mechanical hardness of hearing. Cases which probably belong in this category have been reported by Jackson and Thompson (among others, great diminution of hearing in complete paralysis of the muscles of deglutition, eventually leading to death from starvation).

Foreign Bodies. Traumatic Emphysema.

Foreign bodies of importance are hardly ever encountered in the naso-pharyngeal cavity, apart from the entrance of ingesta in cases of defects of the palate, in dislodgement of the palate by morbid growths, or in paralysis of its muscular apparatus. They can otherwise only gain access by direct introduction, on purpose, or during the act of vomiting. In this manner no doubt an ascaris was conveyed into the nares, which then crept into the Eustachian tube (Andry); and the same is true of a grain of barley, which was found projecting out of the orifice of the tube, in the post-mortem examination of a man who had suffered for several years with an annoying noise in the ear, associated with a peculiar sensation in the lower pharynx (Fleischmann). An old philologist (case of Heckscher), who had been in the daily habit of introducing the Eustachian catheter in his own person, and cleansing his Eustachian tube through it by means of a crow feather fastened to a whalebone probe, happened to get the attachment of this peculiar instrument loosened. The feather remained behind, with one end in the tube and the other in one of the choanæ. After some days the patient freed himself by means of his fore-finger passed up behind the soft palate—a hint towards digital exploration and removal of foreign bodies in similar cases (broken laminaria bougies : Wendt).

The prolonged presence of foreign bodies is not frequent in the lower pharynx either. Such bodies as remain caught in consequence of their size, are usually retained in the region of the cricoid cartilage (where the calibre of the pharynx is slit-formed, and little dilatable). Fishbones, needles, and other sharp bodies sometimes bore themselves into the walls, in various places. More frequently they pass the cavity without exciting injury, or with merely temporary wounding of the mucous membrane, causing a slight amount of pain for some days.

The presence of foreign bodies is usually attended with disturbances of various kinds, pain, difficulty of swallowing, and cough. In a woman, in whom a needle had remained sticking in the posterior wall of the lower pharynx for three years, there was continuous disposition to cough, and "cutting" pain. A chronic catarrh remained after its removal (Hutton). In another case, a palatal plate with six artificial teeth, which was firmly impacted behind the entrance into the larynx, and which had to be removed with polypus forceps, excited an epileptic paroxysm (Matthews). In cases of foreign bodies, of unusual size, filling the lower portion of the pharynx and compressing the larynx, death from suffocation may result: as in a case reported by Bard (a sponge swallowed by an insane patient), and one by Bardeleben (tough veal). A foreign body caused the death of a teacher who, in stooping, broke off the mouth-piece of a long pipe he was smoking. The fragment bored itself into the lateral palatal region without being noticed, according to his statement, and was retained for eight months, giving rise to a tumor on the affected side of the throat beneath the mastoid process. Death ensued in consequence of a hemorrhage from the carotid, which had been wounded by the sharp point of the pipe in a forcible movement of the head (Fingerhuth).

Foreign bodies impacted in the lower pharynx are removed with forceps-like instruments—where necessary, under the guidance of the laryngoscopic mirror. If they are very large, or cause symptoms threatening life, the propriety of making an external opening into the pharynx (subhyoidal) may have to be considered.

Emphysema in the walls of the naso-pharyngeal cavity and the lower pharynx, at first in the immediate vicinity of the Eustachian tube, is not altogether rare, and is, in a certain sense, due to the instrumentality of a foreign body. It follows the use of the Eustachian catheter. The air, which for various reasons may be blown or driven through the catheter, forces its way under the edge of certain little accidental openings in the mucous membrane, and gains access to the cellular tissue. These openings, or losses of substance, may either have existed previously, or may be occasioned by awkward manipulation of the catheter, or by the use of sounds, or of sharp or jagged instruments. The air, having reached the cellular tissue, often spreads rapidly in various directions, generally at once to the corresponding side of the soft palate, to the uvula, and to the lateral and posterior walls of the lower pharynx. The uvula and soft palate often undergo a considerable increase in volume, assume a whitish, spongy appearance (like emphysematous lung tissue), and crackle under the touch. The difficulty of swallowing, and a peculiar alteration in the voice make it almost impossible to overlook the development of emphysema. Left to itself, it may extend over the neck, cheeks, eyelids, even over the entire head, breast, and arms, placing the patient, for a time, in a deplorable condition. I account for the fact that the emphysema, during a certain number of hours or even days, keeps extending to new regions, on the supposition of the existence of a valve-like flap at the point where the air entered, near the orifice of the tube, which opens and shuts in the movements of deglutition.

After a few days, five at furthest, the tumor disappears. With regard to the cause of the sudden death which followed in two cases (Turnbull), after powerful compression of air into the middle ear, with the compression process formerly in use, the view has been repeatedly advanced that it was due to an extension of the emphysema over the mucous membrane of the laryngeal entrance (*lig. ary-epiglottica*), to an emphysema of the glottis, or a compression of the larynx by the tumor in the throat (Voltolini). But other explanations are not to be excluded. At any rate, difficulty of breathing is very infrequent, and only of moderate severity (pressure in the throat), and it no doubt occurs in consequence of the swelling of that portion of the posterior wall lying behind the larynx. In one such case Voltolini saw one ventricular band a little swollen; I myself have seen nothing abnormal in the larynx. At the same time, the possibility of the larynx being involved in the emphysema should always lead to prompt interference.

By *immediate* shallow incisions with scissors into the most accessible parts infiltrated with air (uvula, palatine arches), upon which they usually collapse at once, and by pressing the air out of the subcutaneous tissue with the finger, it is almost always possible to arrest any further development, and to allay the sufferings of the patient. This should not be neglected, even when the commencement of the process has been overlooked, and the emphysema has already spread to a great extent.

A similar submucous emphysema of both cavities, most frequently, and chiefly the upper one, must occur in many cases of fracture of the upper jaw and the body of the sphenoid bone.

An artificial *œdema* of the lower pharynx occurred in a young man, in whom the author attempted to make a copious injection from a syringe through the catheter into the middle ear, so as to remove some viscid secretion through an incision in the tympanic membrane. As he began the operation, the patient at once experienced a sensation as if something ran down to the sternum within the affected side of the throat in the vicinity of the larynx. Swallowing was impeded, and speech was altered as in a case of emphysema, and the lateral cervical region was somewhat sensitive to pressure. The manifestations disappeared during the course of twenty-four hours.

DISEASES

OF THE

STOMACH AND INTESTINES.

LEUBE.

DISEASES OF THE STOMACH AND INTESTINES.

Introduction.

While a thorough knowledge of the anatomy and physiology of the parts involved is a most important pre-requisite to the study of disease in any portion of the body, such information is absolutely indispensable in the investigation of diseases of the stomach and intestines.

It may indeed be asserted that in these affections a knowledge of physiology is the more important, because the products of the organs which are acting under abnormal conditions may themselves be submitted to direct examination. In addition, the essentially chemical nature of the digestive process, as well as the accessibility of the organs concerned, presents the opportunity of obtaining an insight into the nature of the morbid action by experimental methods, and of rectifying, or at least ameliorating, by means of appropriate remedies, the functional disturbances of the stomach and intestines.

For this reason a brief consideration of those points in the anatomy and physiology of these organs, which come most frequently into question at the bedside, shall precede the discussion of the diseases in detail. The object of this introduction being thus fulfilled, the reader may be referred for further information to the various text-books on anatomy and physiology.

AFFECTIONS OF THE STOMACH.

Preliminary Anatomical Remarks.

Position of the Stomach.

According to Luschka, on whose work,[1] which is especially clinical in its scope, the following description of the relative position of the stomach and intestines is chiefly based, full five-sixths of the whole volume of this organ lies on the left side of the body; the remaining sixth, situated to the right of the median line, includes only the pylorus and its surroundings, and lies entirely behind the quadrate lobe of the liver, so that morbid alterations about the pylorus (*e. g.*, ulcers, carcinoma, etc.), can only be palpated when an abnormal subsidence of this part below the border of the liver has taken place. Nor can palpation of the cardia and lesser curvature through the abdominal walls be accomplished, as a glance at the accompanying diagrams, sketched from Luschka's plates, will show.

The vertex of the fundus constitutes the highest point of the stomach, while the lowest point lies in the convexity of the greater curvature, to the left of the median line; to the right of this the curvature rises somewhat abruptly towards the pyloric extremity.

The *positions of the various regions of the stomach are as follows:* The *pylorus* is situated in the right sternal line (drawn down along the right border of the sternum), below the apex of the ensiform process. The *cardia* lies in the left sternal line, above the commencement of the ensiform process. The *lesser curva-*

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[1] Die Lage der Bauchorgane des Menschen. Carlsruhe. 1873.

ture runs near to, and parallel with, the vertebral column on the left side. The *greater curvature*, leaving the border of the liver in the neighborhood of the fundus of the gall-bladder, descends towards the left hypochondrium, within which region the fundus of the stomach appears to be situated *in toto.*

The relations of the stomach to the neighboring parts are interesting, from a pathological point of view. They are as follows: the greater portion of the left segment of the stomach is bounded superiorly by the diaphragm; its fundus rests against the spleen and left kidney; its lesser curvature and a part of the body of the organ are bounded—with only the sac of the omentum intervening—by the pancreas, which lies in a transverse position behind the stomach, and by the accompanying splenic artery and vein; the greater curvature of the stomach and a portion of its anterior surface, as well as its pyloric portion, are bounded by the liver; and, finally, a large portion of its greater curvature rests against the transverse colon. All these organs, which are joined to the stomach by folds of the peritoneum, may, by contiguity, participate with the latter in any morbid process in which this may become involved.

Structure of the Stomach.

The walls of the stomach consist, as is well known, of three layers—the serous, the muscular, and the mucous. Of these, the serous layer requires no further description, since all that is necessary has been said regarding it when speaking of the relations of the stomach to other organs.

The *muscular coat* is directly continuous with that of the œsophagus, and is divided, like this, for the most part, into an outer stratum, the fibres of which run longitudinally, and an inner, in which the fibres run circularly around the lumen. In addition to these two principal strata, there is a third, that of the *fibræ obliquæ*, constituting the innermost stratum.

The structure of the *mucous membrane* demands a fuller description, particularly since our views as to its anatomical conformation have of late become decidedly changed and enlarged.

The mucous layer is connected with the muscular layer of the stomach by a loose, comparatively broad-meshed connective tissue (the stratum cellulosum seu [lamina] nervea). As a result of this arrangement, the mucous layer is enabled, during contraction of the muscular coat, to fall into folds, more or less independ-

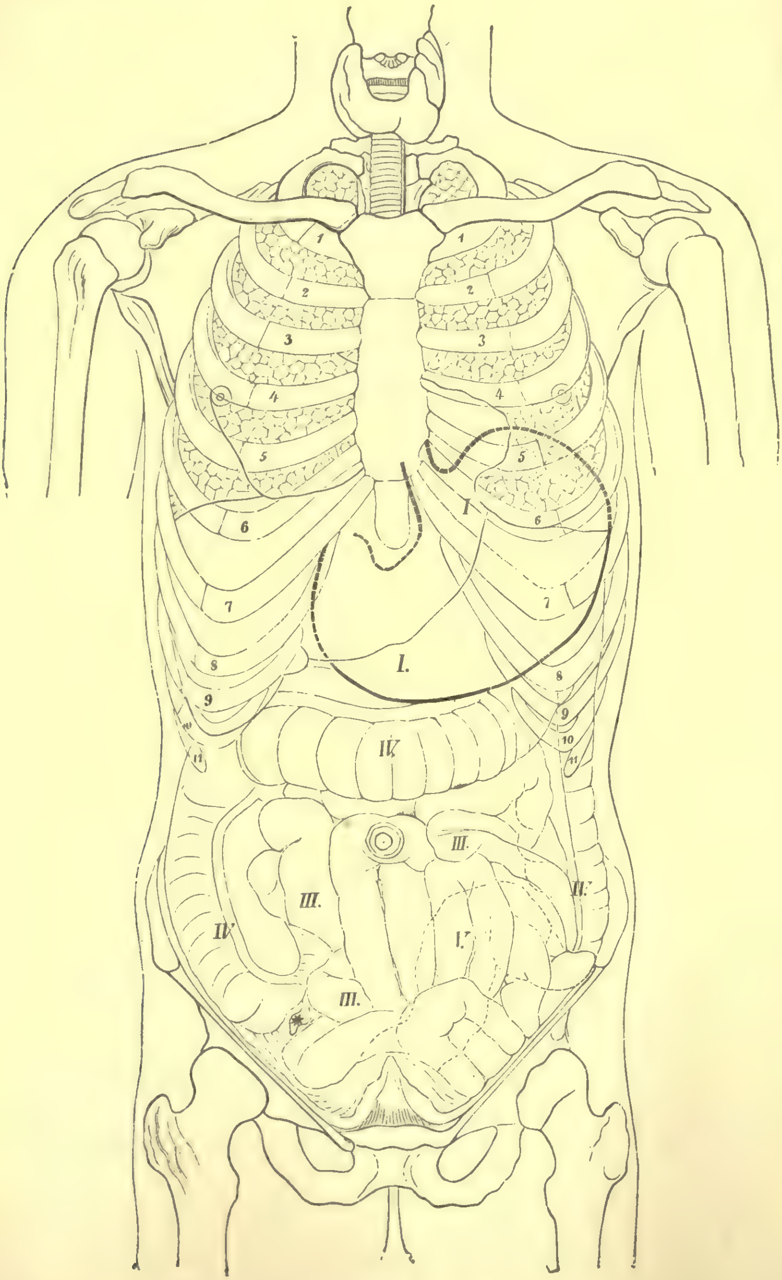

Fig. 1.
Front view of the abdominal organs, according to Luschka.

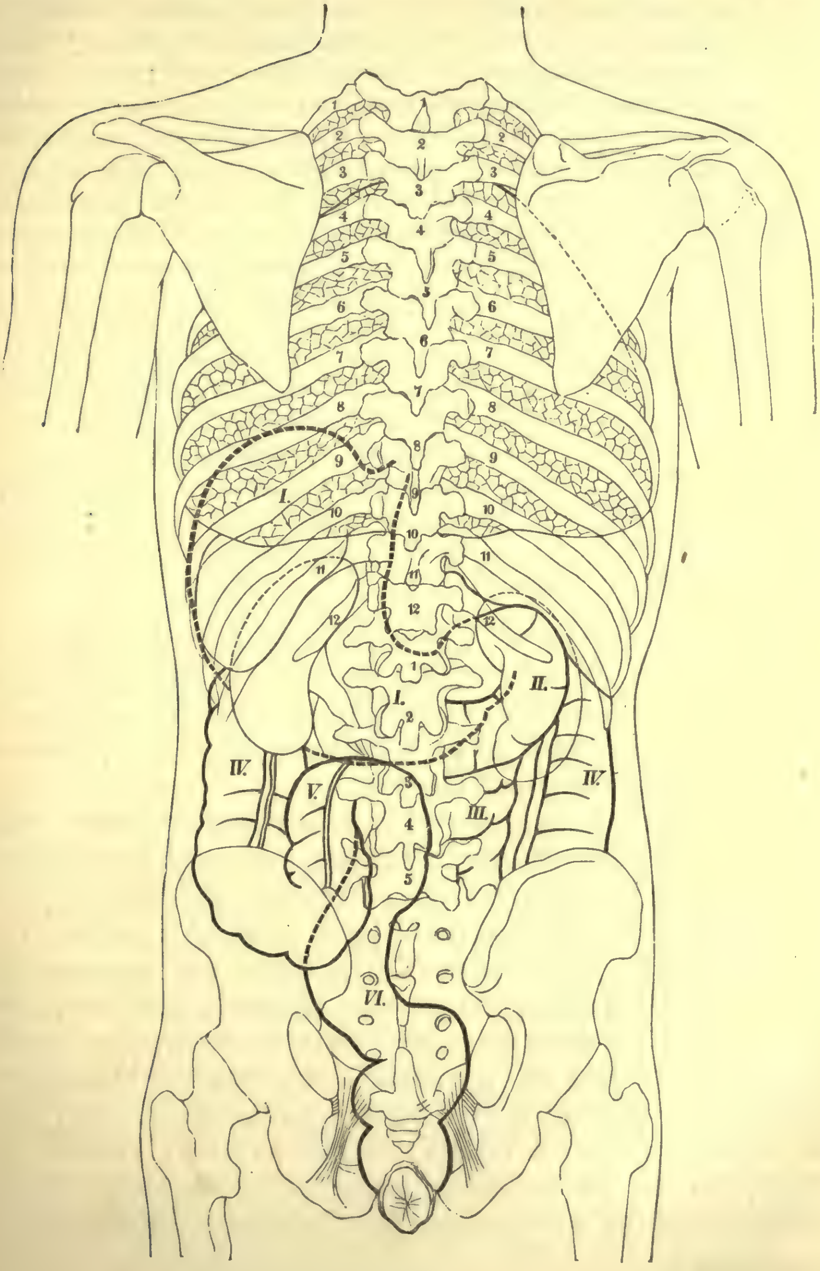

Fig. 2.

View of the abdominal organs from behind, according to Luschka.

I. Stomach. *II.* Duodenum. *III.* Ileum. *IV.* Colon. *V.* Sigmoid flexure. *VI.* Rectum.

ent, as regards their direction, of those of the muscular layer. These folds stand out like large ridges; and, in addition, there is a network of minute wrinkles. The latter give, during life, to the inner surface of the stomach a granular, "warty" appearance (*état mamelonné*), which, however, disappears a short time after death, and which is only met with *post-mortem*, subsequently to the cessation of muscular tonicity, in certain pathological conditions, of which more explicit mention shall be made further on. The relatively considerable area of the mucous membrane, in comparison with that of the contractile muscular layer, may possibly account for the fact that even the sudden tearing-off of a small portion of mucous membrane may occur without considerable loss of blood; a striking instance of which von Ziemssen and I have observed during the use of the stomach pump.[1] It is possible that in such cases the muscular coat, occupying the position where the mucous membrane has been abraded, is greatly irritated by the contents of the stomach, and so becomes roused to energetic contraction, so that the margins of the breach in the mucous membrane become puckered together towards the middle, like the neck of a tobacco-pouch, thus preventing serious hemorrhage.

These small apparent humps, which are created by the natural wrinkling of the mucous membrane, must be carefully discriminated from the permanent hump-like prominences due to the presence of the conglobate glands, which are imbedded in the substance of the mucous membrane.

Next in order to the nervea (passing inwards) comes the *muscularis mucosæ*, which forms the connecting tie between the nervea and the glandular layer proper. This is composed of smooth muscular fibres, which send their processes up between the blind ends of the gland tubules.

The "*propria*," that portion of the mucous membrane which was first accurately described by Henle, and which is situated in immediate contact with the muscularis mucosæ, seems to serve as a framework for the innermost layer of gland tubules. It is essentially of the nature of connective tissue, and possesses two varieties of cell elements of somewhat uncertain significance—the *stellate cells*, belonging more particularly to the true wall of the gland tubule; and the *lymphoid* cells, situated between the gland tubules, and often existing in great numbers. The latter are crowded together in certain localities to such an extent that they press the gastric glands aside, and form lenticular protuberances of the mucous membrane (glandulæ lenticulares seu conglobatæ).

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[1] *v. Ziemssen*, Deutsches Archiv für klin. Med. X. pp. 68–69.

Their number varies considerably in different individuals; occasionally they are altogether wanting; at other times they form groups, scattered over the entire stomach. Their peculiar deportment in typhoid fever is interesting. In this affection they occasionally become ulcerated, like the agminated glands of the small intestines, of which I have recently had an opportunity of observing a striking example.[1]

Finally, the true *gland-layer* presents itself as the innermost part of the mucous membrane. The glands of the stomach, estimated at five million in number, belong to the tubular variety. Two sorts have long been distinguished, the "*peptic*" glands and the *mucous* glands. These designations connect with the names the supposition of certain functions for both glands. On account of the variations in our physiological opinions, which, on this subject especially, have of late become exceedingly confused, it will be advisable to distinguish both kinds of glands only by their distribution and anatomical relationships, and, therefore, to speak of "*principal glands*" (Hauptdrüsen) and "*pyloric glands.*"

As regards the *microscopic* character of the contents of the glands, recent investigations by Rollett and Heidenhain have decidedly modified the views formerly held.

The *pyloric glands* are found in the neighborhood of the pylorus, and are more simple in structure than the principal glands; the columnar epithelium, which is found around the openings of all the glands, extending down in the former continuously to the very bottom of the gland, only displaying more diminutive and finely granular cells at the lowest part.

The *principal glands*, on the other hand, contain, in addition to columnar epithelium, large, dark, flat, granular *lining cells* (Heidenhain; formerly called "peptic cells"), which are found most plentifully at the commencement of the gland tubule. To particularize: after the initial portion (*Anfangsstück*)[2] of the gland, with its slender columnar epithelium, follows a boundary portion with somewhat wider, more granular cylindrical cells; next to this comes a portion (the "*middle portion*") in which the columnar epithelial cells become so insignificant in comparison with the lining cells, that certain observers (Rollett, Frey, and others) altogether deny the presence of other than lining cells in this section of the gland. So far as I can judge from my own preparations, I must, as regards this point, share the views of the latter authorities; yet Henle may be correct in asserting that the truth lies midway—that columnar epithelial cells are sometimes wanting entirely in this neighborhood, and sometimes are present, but are crowded aside by the lining cells. Finally follows the true gland tubule (the *terminal portion*), which, as is generally acknowledged, contains both kinds of cells; the cylindrical cells lie toward the lumen, so that this appears to be entirely bounded by columnar epithelium, while the lining cells are in contact with the wall of the tubule, and not unfrequently protrude outwardly. In certain cases one or the other kind of epithelium appears to predominate in the human stomach, so that in these instances, as Henle states, sometimes only a few flat cells are present; at other times both the middle portion and the blind extremity appear for the most part filled with flat, granular lining cells. (See illustrations in Henle's "Eingeweidelehre," 1873, p. 169.)

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[1] Klinische Berichte von der medicinischen Abtheilung des Landeskrankenhauses zu Jena, herausgegeben mit *Penzoldt* von *W. Leube.* Erlangen. 1875.

[2] The most convenient designation is that of *initial portion* (Magengrübchen), *middle portion* (neck of the gland, external intermediate portion, Rollett), and *terminal portion* (body of the gland).

In addition to these specific glands, there are also usually, according to Cobelli, certain racemose mucous glands in the region of the pylorus.

The surface of the mucous membrane appears *irregularly* provided with villous projections between which the openings of the peptic glands are situated.

The *vascular supply* is arranged, in accordance with the almost invariably tubular shape of the glands, in the form of long-meshed networks, which are so disposed that, from the arterial twigs in the nervea, very delicate vascular canaliculi run parallel with each other to the surface of the mucous membrane, and surround the wall of the gland with a network of capillaries. At the openings of the glands these form roundish plexuses, which consist of larger vessels, and which should probably be regarded as the commencements of veins. The elongated venous radicles, proceeding from this point by a pretty straight course, increase rapidly in volume, and are much more scattered than the ascending arterial twigs, so that Frey conjectures that a certain amount of resistance is thus offered to the return flow of blood in them.[1]

The *lymph canals* of the gastric mucous membrane extend, as Lovén has recently proved by injected preparations, to the very surface of this membrane, not, as was formerly supposed, merely to the blind ends of the gland tubules. This is, of course, important in connection with our ideas as to the means by which absorption takes place through the gastric mucous membrane.

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[1] See *Henle's* "Eingeweidelehre," 1873, Fig. 119, and Fig. 466 in *Frey's* Histology (Eng. trans.).

The *nerves* originate from the abdominal portion of the vagus, which, on the left side of the body, is distributed almost exclusively to the stomach, and in fact to its anterior face, after having formed the anterior gastric plexus upon the cardia. The right branch, on the other hand, sends only one-third of its filaments to the gastric parietes, more particularly to the posterior, while the remaining two-thirds are distributed to the other abdominal organs. The branches of the vagus form anastomoses with those of the sympathetic, which, proceeding from the cœliac plexus, sends a number of ramifications, in company with the larger vessels, to the stomach. The nerve filaments arising from this double source form in the submucous layer a network which is provided with numerous small ganglia.

Preliminary Physiological Remarks.

Until the middle of the last century the ideas prevalent regarding the process of *digestion* were somewhat vague. Thus mention was made of coction, fermentation, decomposition, or maceration of the food taken into the stomach. It is true that Boerhave[1] had already somewhat restricted the theory of fermentation in the stomach by calling attention to the part played as associated causes of digestion by the mechanical action of the muscular structure of the stomach upon its contents, and by the movements of the neighboring great vessels, of the diaphragm, and of the muscular structure of the abdomen generally. But, in the period immediately following, the theory of fermentation received support, apparently exact and experimental, from the researches of two Englishmen, Pringle and McBride,[2] who carried out the idea, subsequently proving so fruitful, of imitating digestion artificially, outside of the body. They showed that, at the proper temperature, food, mixed with saliva, proceeded to a sort of fermentation with the development of gas, and they inferred the occurrence of a similar transformation of the food, mingled with saliva and gastric juice, within the stomach. The researches of these two experimenters were, however, by no means exact. Those of Réaumur and Spallanzani were the first which were conducted with that thoroughness and precision which physiological investigation demands. While the former showed that the fluid formed in the stomach is capable of digesting fragments of meat, Spallanzani, incomparable as a physiological investigator, proved, in opposition to previously received views, that the gastric juice hinders fermentation in the stomach, since putrefying meat, after remaining in this organ a certain time, becomes deprived of its bad odor. Spallanzani instituted upon himself the highly ingenious experiment, which had already been performed, of swallowing small wooden tubes, pierced with holes, so as to allow free access to the fluids of the stomach. The result showed that it is the gastric juice, and not, as is the case with certain birds, the movements of the muscular structure, which brings about digestion of the food ; for while the wooden tubes swallowed escaped digestion, the enclosed meat did not.

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[1] Instit. med. Physiol. § 76–89.

[2] *Spallanzani*, Researches upon the Gastric Juice. Part VI.

From the period of these pioneers' experiments our knowledge of gastric digestion has developed step by step upon the firm basis of physiological experiment. The most important discoveries of the period immediately succeeding that of Spallanzani, are : the proof of the existence of free hydrochloric acid in the gastric mucus by Prout in 1824, and the demonstration of pepsin by Schwann in 1836. The study of normal digestion was greatly aided by a fortunate accident, which brought under the notice of Beaumont a man suffering from gastric fistula, the result of a gunshot wound. Still further aid was gained through the artificial production of active gastric mucus by Eberle, and, finally, by the establishment of gastric fistulas in living dogs. Thus were obtained the means requisite for a clear understanding of the chemical processes concerned in digestion.

The digestive influence of the stomach upon the ingesta is due, in the first place, to the *gastric juice*, the specific secretion of the gastric glands. This is *acid* in reaction, and is only found in the stomach as a result of the action of some irritant upon the mucous membrane,—the mucous surface of the empty stomach appearing barely moist, and yielding no decided reaction. Secretion once aroused, the excitation easily extends from the locality originally stimulated to the neighborhood. The gastric juice may indeed continue to be poured out even after the irritation which originally excited it has been withdrawn, as, for instance, after the alkaline saliva swallowed has been neutralized, or after the ingestion of solution of soda which gives rise to a particularly persistent secretion.

The acid reaction of the pure gastric juice, obtained by mechanical irritation of the mucous membrane of the empty stomach, is dependent upon the presence of an *acid*, the nature of which has been a subject of discussion for some decades past. After Prout, in 1824, had shown that the gastric juice affords hydrochloric acid on distillation, direct proof as to the presence of free hydrochloric acid in this secretion was brought forward by C. Schmidt. This investigator demonstrated, by quantitative analysis of the ash of gastric juice, that the amount of hydrochloric acid precipitated by nitrate of silver was more than equivalent to the proportion of bases present. The amount of free acid present is, according to several analyses, about two-tenths of one per cent.

Frerichs, on the other hand, had already, in his well-known and valuable work upon digestion, declared himself in favor of *lactic acid* as the normal acid of the stomach. This view, originally brought forward by French investigators, has continued dominant in France up to the present time.[1]

Although, according to researches which have been thus far made, it must be admitted that hydrochloric acid is the true acid of the gastric juice, yet, on the other hand, the important practical fact must not be forgotten that after the stomach has become filled with food no free hydrochloric acid can be found in it. On the contrary, by the transformation of its contents during the act of digestion, other acids are set free, in particular lactic acid (by the metamorphosis of starch in the stomach), and these, as well as butyric acid, are supplanted in the salts containing them by hydrochloric acid. While these acids most probably originate from the food, the hydrochloric acid may be looked upon as an actual product of secretion on the part of the gastric mucous membrane.

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[1] See the latest edition of *Béclard's* widely-read Physiology.

The second principal constituent of the gastric juice, in addition to the free acids, is an albumen-dissolving, so-called hydrolytic ferment—*pepsin.* This substance, in conjunction with hydrochloric acid, exerts a solvent action upon albuminous matters, changing the latter into peptones, which represent chemical and physical modifications of the albuminates. Neither pepsin nor hydrochloric acid singly (although the latter, after long action, seems to exert a peptonizing influence) is capable of affecting that transformation of albuminous substances which is observed in digestion. Both constituents must, in fact, be present in the gastric juice in a *certain relative proportion* in order to induce energetic digestive action.

In order that a gastric fluid shall retain any considerable digestive power, the *proportion of acid* contained must not vary from the normal except within very narrow limits. Digestive fluids whose proportion of acid falls short of or exceeds these limits digest poorly; but since different albuminous substances require different quantities of acid for their rapid solution, no fixed grade of acidity can be specified as most favorable under all circumstances. Thus, for example, gluten appears to be most easily dissolved when the proportion of acid is one per cent., while boiled albumen requires only 0.15 per cent. In general, it may be stated that the proportion of hydrochloric acid in the digestive fluid should not amount to more than seven per cent. in order that it may exercise the most energetic digestive influence.

Not only the amount, however, but also the *nature* of the acid is of moment with respect to this object. To be sure, the various acids, acetic, oxalic, lactic, phosphoric, etc., form active mixtures in combination with pepsin; but larger quantities of these acids must be employed in all cases, if an effect comparable to that produced by the hydrochloric acid mixture is desired,—in the case of lactic acid, for instance, more than ten times as much.

As regards the other principal component of the gastric juice—pepsin, in accordance with its character as a ferment—the smallest quantities are sufficient, when mixed with dilute acids, to dissolve albuminous bodies. Thus, it has been shown that pepsin, in the proportion of one part to 60,000 of fluid, is capable of fulfill-ing this object perfectly. *Yet, up to a certain point, the rapidity with which the conversion into peptones proceeds is proportionate to the quantity of pepsin present in the digestive fluid.* Beyond this maximum, however, the presence of more pepsin is superfluous.

Most important for the comprehension of the digestive process under both physiological and pathological conditions is this fact—namely, that in experiments upon artificial digestion, dissolved albuminous matters become completely changed into peptones only when the latter, as they are formed by the action of acid and pepsin upon albumen, are continually removed by dialysis. *The accumulation of peptones in the digestive tract is thus an obstacle to the further solution of those portions of the albuminous materials not yet peptonized.* So soon, however, as the completed products of digestion have been separated from the latter by diffusion, fresh portions of dilute acid can initiate once more the solution of the albumen. The pepsin, in spite of its digestive action, itself remains apparently unaltered, and does not lose in strength. This peculiar action of pepsin in association with hydrochloric acid upon albuminous matters has been explained (by C. Schmidt) as the effect of a compound acid, pepsin-hydrochloric acid, which effects the transformation of albumen. In the process of digestion the hydrochloric acid separates from the pepsin-hydrochloric, combines with the albuminous matters, and while in the nascent state changes these into peptones.

Meanwhile the pepsin, set free by this change, repeats, with a fresh relay of hydrochloric acid, the process of transformation. The gelatigenous tissues, like the albuminous, are also dissolved by the gastric juice with the formation of peptones.

The *formation of peptones* goes on slowly, permitting several stages to be distinguished, each of which is characterized by the presence of certain peculiar products of transformation in the digestive tract. Thus, at the beginning of the digestion of fibrin, a product, *parapeptone*, identical with syntonin, is formed. This is a modification of albumen, and may be formed, although somewhat slowly, externally to the organization by the action of hydrochoric acid upon albuminous matters. In acid solution it does not coagulate upon boiling, but it is thrown down when the solution is neutralized by alkalies. The changes taking place during the digestion of albuminous substances do not stop at this stage, but progress to the formation of true *peptones*, not coagulable upon boiling, not thrown down by either acids or alkalies, and diffusing themselves with ease. The osmotic equivalent of these products is, in any case, seventy times lower than that of ordinary albumen, which scarcely diffuses itself at all through membrane against water. For this reason the simplest method of separating peptone from albumen is to place the mixture upon a membrane of vegetable parchment. Brücke notes the following distinguishing characteristic between peptones and albuminous bodies: *Albuminous bodies are precipitated from acid solutions by ferrocyanide of potassium; peptones are not.*

The gastric juice is formed in the mucous membrane of the stomach. As a result of Heidenhain's researches (vide Anat.), the view has recently gained favor that the "principal cells" of the gastric glands are concerned with the formation of pepsin, while the "lining cells" secrete the hydrochloric acid. Ebstein, in consideration of the similarity of structure between the principal cells of the principal glands and the cells of the pyloric glands, has attributed the formation of pepsin also to the latter, and has given to these the name of peptic glands. Various researches upon this subject, made thus far, offer no support to Ebstein's view, but rather tend to confirm the theory, now prevalent for some time, that the formation of pepsin and hydrochloric acid goes on in the principal glands of the gastric mucous membrane, while the pyloric glands secrete a mucous fluid without digestive power.

Confirmatory of this view, we find that while the mucous membrane of the pyloric region in a recently killed animal gives a weak alkaline reaction, the mucous surface in the region of the principal glands changes the color of blue litmus paper to red.

The acid reaction of these parts of the mucous membrane is confined, however, to the opening of the gland, while the fundus of the latter again yields an alkaline reaction. Moreover, this acid reaction of the surface appears to present itself *only during* the activity of the glands, and the apparent contradiction to this, shown by the acid reaction of the surface of an *empty* stomach, may be explained by the fact that the saliva swallowed just before death may have excited the glandular secretion. If the ingestion of saliva be prevented, by strangling the animal, the free surface of the stomach never affords an acid reaction (Rollett).

These physiological experiments serve to answer the question as to the possibility of *self-digestion* on the part of the stomach, a subject which has been much discussed, particularly in connection with the pathogenesis of gastromalacia. As has been said, only the surface of the gastric mucous membrane affords an acid reaction; while in the cadaver, not only *all* the layers of the mucosa, but also the muscularis and serosa are acid. This is to be regarded as a purely post-mortem phenomenon, and since we can only conceive a digestive process as going on in the stomach when the reaction is acid, so, logically speaking, digestion of the superficial coats alone of the stomach could be regarded as occurring during life. The appearance of cell-debris (according to Heidenhain, of cylinder cells, not of lining cells) from the exits of the gland tubules in the mucous layer covering the surface of the mucosa, as well as the constant presence of peptones in normal gastric juice, favor this view. Whether this self-digestion, which occurs under normal circumstances, may under abnormal influences extend more deeply into the tissues of the gastric walls, and under what conditions the same can occur, will be further spoken of under the head of Gastromalacia and Round Ulcer of the Stomach.

The amount of gastric juice daily poured out is considerable. It may be estimated to amount to as much as thirty pounds per diem, of which the larger amount is again absorbed. The same liberal estimate has been made regarding the absolute *potentiality* of the gastric mucous membrane. Schiff has calculated that a sufficiently dilute infusion of the gastric mucous membrane of a dog in good physiological condition is capable of digesting two hundred pounds of albumen!

The *production of gastric juice is favored* by the most various circumstances. Foremost among these are mechanical irritation of the mucous membrane, and the presence of solid food, not too well chewed, which may be regarded as an energetic excitant: similarly, alkaline fluids, *saliva*, rapid changes of temperature, as in the ingestion of cold water, all act in exciting the production of this digestive fluid. On the other hand, *the activity of the gastric juice is suspended*, or at least weakened by the presence of substances in the stomach which interfere with the progress of fermentation, and particularly suspend the digestive power of the pepsin. Such substances are, the metallic salts, concentrated alcohol, concentrated acids, etc. In addition, Bernard has shown that *acid, as well as simple, solutions of peptones are precipitated by bile—the pepsin being thrown down at the same time as the albuminous bodies.* The gastric juice is thus robbed of its pepsin, so that the bile exercises a hindering influence upon the activity of the gastric juice similar to that of the chemical reagents mentioned.

These precipitations by means of bile are probably dependent upon the fact that the glykocholic acid which is thrown down by the acid gastric juice carries the pepsin ferment and the peptones with it mechanically, a reaction which may be taken advantage of for the isolation of other ferments. Since the precipitate of glykocholic acid and pepsin is again dissolved by hydrochloric acid (Burkart) and once more assumes digestive properties, while the albuminous precipitate is soluble in weak alkalies and especially in the intestinal juice, the following important conclusions may be drawn. In the first place, the entrance of bile into the stomach, not unfrequently observed in certain diseases, may disturb digestion by precipitating pepsin from the gastric juice. Subsequently, however, a fresh secretion of acid gastric juice may dissolve this precipitate anew, set free the pepsin, and rectify the difficulty. In the second place, it is worthy of particular notice that the albuminous materials which reach the intestine, altered by the gastric juice but not yet absorbed, are precipitated in the upper portion of the gut and remain clinging to its walls until they are gradually dissolved by the intestinal juice, and are finally absorbed (Kühne).

Although direct proof was given by Spallanzani that digestion in the human stomach is an essentially chemical process, and that mechanical trituration of the food, such as occurs in the stomachs of birds, does not take place here, yet we must not on this account underrate the aid which is afforded by the gastric *muscular structure* in the process of digestion. To appreciate this, we need only consider how greatly we can hasten the effect of an artificial digestive fluid upon the substance to be dissolved, by agitating the vessel in which the mixture is contained. Through contraction of the walls of the stomach, the different portions of the chyme are, it is plain, brought into intimate contact with the same, and thus become saturated with gastric juice. Moreover, this same contact of the gastric contents with the walls of the organ tends constantly to excite further secretion through mechanical action. Furthermore, the movements of the stomach aid in the formation of peptones, as well as in the absorption of those already produced. For after what has been said previously with respect to the retarding influence of accumulating peptones upon the progress of digestion by pepsin, it is easy to understand that, if the peptones formed on the periphery of the bolus are not immediately removed, the peptonizing process, so far as the interior is concerned, must come to a stand-still. The movements of the muscular structure aid this removal, and consequently, in my opinion, the absorption of the peptones. They do this because the muscular contractions render the lymph and blood currents more active, and consequently increase the absorption of peptones by the lymphatic radicles which stretch up between the glands (similarly as in the intestinal villi; see farther on).

The mechanism of the special movements of the stomach is as yet not perfectly understood. It appears pretty certain, however, that the orifices of the stomach are more or less closely shut (the pyloric by reflex action) when this organ is full, and that the gastric walls are firmly contracted about their contents, so as to render certain movements evident, those, namely, which take place peristaltically from the fundus toward the pylorus. Finally, the stomach undergoes a movement of rotation on its long axis independently of the movements of muscular contraction, so that the greater curvature rises from below and is directed anteriorly. From time to time the organ contracts more strongly, and the chyme is emptied into the small intestine through the temporarily open pylorus. The *cause of this expulsion* is probably to be found in irritation of the walls of the stomach by the increasing acidity which occurs towards the end of digestion (Brücke).

Imperfectly as the details of muscular movement in the stomach are understood, still less is positively established regarding the influence of the nervous system upon this movement and upon the production of gastric juice. As regards the former, it seems most probable that the movements of the stomach proceed from the ganglia situated in the parietes, and that these themselves are influenced by the vagus, and perhaps (as Goltz's experiments upon the stomach of the frog would seem to show) by the central nervous system, the action of which is inhibitory. Thus the separation of the ganglionic apparatus from this system results in a great increase in irritability on the part of the ganglia. In the same manner the process of digestion itself appears to heighten the irritability of the nervous elements.

Finally, the influence of the nervous system upon the secretion of gastric juice is still more obscure, though this, like the secretions of other glands, is essentially the effect of the excitation of certain nerves. Irritation or section of the vagi, irritation of the splanchnic, etc., afford no constant result in altering the secretion of gastric juice.

While the albuminous substances, influenced by the peculiar gastric glandular secretion, undergo those metamorphoses which have been described, the other constituents of the food remain more passively in the stomach. The *fats* continue, for the most part at least, unaltered, and pass through the pylorus as such, or in rare cases after the separation of the fatty acids. The transformation of the *carbohydrates*, and in particular of the cooked starch, continues, even without the aid of the gastric juice. In the early period of gastric digestion the mucus continues to effect the change of starch into dextrine and sugar. Later, lactic acid fermentation takes place, during which the starch, probably under the influence of a ferment originating externally to the stomach, is changed into dextrine and lactic acid. Glucose, the intermediate product of this process, is quickly transformed, and is only found among the contents of the stomach in small quantity.

The gastric contents, taken as a whole, form an acid *pap*, which is called *chyme*, and consists in part of unaltered food and in part of those portions of the ingesta which have been transformed by the digestive fluids of the upper portion of the alimentary canal. Thus we find in the chyme unaltered albumen, parapeptone, peptone, fat, occasionally fatty acids, lactic acid, and glucose. This last substance is formed in part from starch, and in part probably, according to Hoppe, from cane sugar, by the action of the gastric mucus. In addition to the substances above mentioned, certain gases, composed of oxygen, nitrogen, and carbonic acid, are also found in the stomach. The last named collects under normal conditions, because the oxygen contained in the air swallowed is absorbed from the stomach into the blood, and to take its place carbonic acid diffuses itself from the blood into the stomach. When, however, abnormal changes take place in this organ, as happens in disease, and occasionally even in health, other gases, as hydrogen, hydrosulphuric acid, etc., are formed, to which further allusion will be made in treating of the various diseases of the stomach.

While the chyme usually passes on through the pylorus, yet, under certain circumstances, as in vomiting, a reverse peristaltic movement of the gastric contents takes place. *Vomiting*, however, is such an important symptom in many diseases of the stomach, that the mechanism of its occurrence demands especial description.

The *act of vomiting*, by which, as is known, the contents of the stomach, together with those of the œsophagus and the upper part of the intestines, are, under certain circumstances, emptied upwards and outwards, comprises the following separate phases: restlessness, lassitude, vertigo, swimming in the head, violent perspiration, increase of saliva, nausea, and gulping. By means of this last movement, air is introduced into the stomach, and, in certain individuals, inflation of the epigastrium results. Then occur retching, a deep inspiration, and, immediately following, a forced expiratory effort, whereby the contents of the stomach are driven outwards through the œsophagus and mouth.

The mechanism of vomiting is not yet entirely understood in all its particulars; still, we possess, in works already published upon the subject, material for a better understanding of this complicated process.

The *nausea* which precedes the act of vomiting is a muscular sympathy, excited by anomalous movements of the pharyngeal and palatal muscles, and these movements are brought about, through reflex action, by certain irritations of the gastric mucous membrane. The involuntary *gulping*, which follows, introduces air and saliva into the stomach, and brings about, as before mentioned, that inflation of the epigastrium which takes place just previous to the vomiting. Entrance of air into the stomach is also favored by the now increased *retching*.

Retching must be regarded as incomplete vomiting. Like the latter, it is introduced by an inspiratory effort, together with, apparently, a closure of the glottis; the latter movement prevents an equalization of the atmospheric pressure, by stopping all entrance of air into the respiratory passages. Under these circumstances, the air, streaming in, under the influence of the inspiratory effort, through nose and mouth, finds its way into the upper part of the œsophagus in considerable volume, opportunity being offered by the depression of the larynx which takes place in forced inspiration. If the inspiratory efforts made during retching are followed by attempts at swallowing, the air contained in the entrance of the œsophagus is drawn into the stomach, the inflation of which is thus furthered. For the same reason a portion of the gastric contents must be drawn into the lower end of the œsophagus, provided the cardia be patulous, a condition which the abdominal pressure would actively aid in bringing about.

After the efforts at retching have continued for a longer or shorter period, a deep inspiration, with simultaneous closure of the glottis, takes place, instantly succeeded by a strong expiratory effort, with specially energetic contraction of the abdominal muscles. Meanwhile, as a result of these complicated respiratory movements, suction of the gastric contents into the lower portion of the œsophagus takes place, on the one hand, while, on the other, the stomach *is pressed together by the simultaneous contractions of the diaphragm and the abdominal muscles.* The contents of the stomach are thus forced upwards, and are passed along through the œsophagus over the closed glottis to the mouth. It is taken for granted that the cardia is open and the pylorus closed, which conditions are fulfilled *by the stomach itself taking an active part in the act of vomiting.* Schiff has proved experimentally that the opening of the cardia is due to such action on the part of the stomach. Further, since the stomach can only be perfectly emptied through the cardia when closure of the pylorus prevents escape of the gastric contents into the intestine, it follows that during vomiting this orifice remains firmly shut.

The closure of the gastric outlet is only temporary, since, after vomiting has continued some time, bile passes from the duodenum into the stomach. The cause of this influx may probably be found in a relaxation on the part of the abdominal muscles, which takes place after long-continued vomiting, allowing the hitherto compressed stomach to expand and draw in bile.

The *œsophagus does not take an active part* in the ejection of chyme from the stomach,—indeed, its muscular structure remains completely relaxed, as Wild and Ludwig have shown, disproving thus the idea of "antiperistaltic movements of the œsophagus" in vomiting.

The *causes* of vomiting are very numerous, but the immediate incitement to this act may always be ascribed to irritation of the nerves with which the act is directly connected. We are not as yet in a position to affirm precisely by what course this irritation is conveyed. Excitation of the nerves connected with vomiting may take place in the most various ways, central or peripheral, direct or reflex. Thus, vomiting is observed in diseases of the brain, in affections of the pharynx, œsophagus, and stomach—as a result of irritation of organs connected with the peritoneum, such as the uterus, the kidneys, etc.

After all, what interests us most is, that *the cause of vomiting commonly lies in the stomach itself, and that it occurs most frequently in those diseases which affect the orifices of this organ.* This clinical fact is in accord with what has been said regarding the mechanism of vomiting, and also with the results of experiment; it has long been known (Beaumont) that touching the mucous membrane of the human stomach, through a fistula, with the finger, arouses the inclination to vomit. Hermann has shown recently that tartar emetic—*the* emetic, *par excellence*—acts more quickly and energetically when taken by the mouth than when injected into the veins, in which case it only comes in contact with the parietes of the stomach indirectly.

Gastritis.

Inflammation of the Stomach.

(Gastric catarrh, Inflammatory dyspepsia (Todd), Embarras gastrique, Magenentzündung.)

The older literature will be found in *Plouquet's* literat. medic. digest. 1809, under the head of Ventriculi inflammatio.—*Lieutaud.* Histor. anat. med. 1767.—*Broussais*, Histoire des phlegmasies, etc. 1822.—*Louis*, Du ramollissement, etc. Archiv. génér. de médec. Tome V. Mai 1824. p. 1 et seq.—*Andral* (John), Abhdlg. über die anat. Kennzeichen der chron. Magenentz. Répert. génér. d'anat. etc. To. I. Kap. 1. 1826. Samml. auserl. Abhdlg. 33 S. 702.—*Annesley, J.*, On Inflammations, etc. in his Researches, etc. 1828. Sammlung auserl. Abhdlg. 35. S. 551.—*Abercrombie*, Diseases of the stomach. German translation by Gerhard v. d. Busch. 1830. S. 15.—*Naumann*, Handbuch der medicinischen

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Acute Gastritis.

Inflammation of the mucous membrane of the stomach (acute) was evidently recognized by the older physicians only in those cases where marked changes had taken place in the gastric parietes. Thus, although Celsus[1] speaks of an "*inflammatio ventriculi*," his description shows it to be a different affection from that now called gastritis, since he regards it as rare, and describes a "*resolutio stomachi*," with attendant disorders of nutrition, as the commonest among diseases of the stomach and as quite distinct from the former. It may easily be perceived, from Boerhave's description of "*inflammatio ventriculi*" (Aphorism, § 951), and the commentary on it by Van Swieten, that at that time only the severest forms of gastritis, and in particular those induced by toxic influences, were classed as inflammations of the stomach.

Boerhave, however, had already observed very correctly that the stomach may be the seat of inflammation just as any other organ, and that the *exacerbations of pain in the gastric region observed under these circumstances are due to the ingestion of food.*

The most notable advance in generalizing the theory of inflammation of the stomach was made at the beginning of the present century by Broussais, who, with the revolutionary originality peculiar to himself, designated "gastro-enteritis" as the basis of the most various diseases, and gave immoderate extension to its influence throughout all pathology.

Although subsequently gastritis came to be limited within narrower bounds, it has still remained one of the commonest diseases, its literature being exceedingly copious,[2] particularly if works on "*dyspepsia*" be included.

The latter designation, "*difficult digestion*," does not indicate an independent disease, but only a group of symptoms common to the most various affections of the stomach, and which must be regarded as significant of abnormal processes of digestion in this organ. To be sure, the latter may exist without giving rise to symptoms of dyspepsia—capricious appetite, eructation, nausea, etc.[1] This, however, does not justify the theory of a purely functional dyspepsia without anatomico-pathological changes of the gastric mucous membrane, for in these very cases, as Beaumont (l. c. p. 100) observed in his patient, before *any disturbance of the appetite occurred*, the gastric fluids became "acrid," and the edges of the fistula eroded, while red, even "aphthous" (?) patches appeared on the surface of the mucous membrane, together with other evident signs of disease in the mucosa. I shall, therefore, not devote a separate chapter to the description of "*dyspepsia*," but shall allude to its origin, the symptoms produced by it, and the therapeutics necessary for its relief, when speaking of the different diseases of the stomach.

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[1] De Medicina. Lib. 4. Cap. 5.

[2] The bibliography given above only includes the more important works of which I have made use.

Pathological Anatomy.

Acute catarrh of the stomach, in its less severe forms, leaves only slight traces post-mortem. The significance of these alterations should be interpreted with great caution, because anatomical changes, due to its specific contents, take place in the stomach after death, changes which may be regarded as the result of self-digestion of the inner coats of this organ. In marked cases we find injection and infiltration of the mucous membrane, to a greater or less extent, confined, however, mainly to the region about the pylorus, sometimes with, sometimes without ecchymosis. The surface is covered with a tough, somewhat adhesive layer of mucus, occasionally containing traces of blood. These naked-eye appearances are observed, not only in the cadaver, but also in the living subject. Beaumont, who had, as is known, the opportunity of observing the condition of the surface of the stomach through a fistulous opening in the case of the Canadian, St. Martin, describes its appearance, during an attack of catarrh, briefly as follows: "The mucous membrane is sometimes red and dry, at others pale and moist. The secretion of gastric juice is diminished, occasionally suppressed entirely. In a few instances irregular red patches are observed, from one-half to one and one-half inches in circumference, covered now and then with aphthous crusts.'' Beaumont also speaks of conditions resembling the rolling up of the mucous membrane into small folds and strings, and of pustules on the mucosa, appearances which conflict somewhat with our present views as to the anatomical changes in the gastric mucous membrane.

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[1] I have proved the correctness of this proposition by passing a suitable tube into the healthy human stomach, from eight to ten hours after a meal. The organ is almost invariably found empty, with the exception of certain acid malodorous contents, which may be present without the individual feeling unwell.

The anatomical changes alluded to correspond to those observed in the mucous membrane of animals in which gastric catarrh has been artificially induced (*e. g.*, by alcohol). *Microscopic* investigation of the mucous membrane, thus inflamed, gives results which are of especial value, because the tissues are examined immediately after the death of the animal before self-digestion can take place. In such examinations Ebstein found *no change in the lining cells, while the principal cells showed persistent cloudiness, more marked granulation and shrinking, and occasionally complete fatty degeneration.* These last alterations, as well as the *long resistance to change after continued irritation*, are thought to be pathognomonic of inflammation, as distinguished from the alterations which take place in the epithelial cells during digestion, and are, in other respects, essentially the same as in inflammation.

Etiology.

We have accustomed ourselves, in part through reaction from the teachings of Broussais, in part under the influence of the prevailing theories of pathological anatomy, to place transitory dyspepsia in the category of gastric catarrh, and to look without further thought upon influences which give rise to dyspepsia as causes of gastritis. If I conform to this custom in the following pages, it is because I regard it, for the reasons given above, as, for the time being, correct. I am well aware, however, that exact anatomico-pathological, that is, experimental, proof of the mutual relations existing between the digestive changes about to be described and the inflammatory alterations in the gastric mucous membrane has yet to be given. Simple acute gastric catarrh is a disease which terminates so rapidly in recovery that the difficulty of obtaining a demonstration of this kind must be evident. On the other hand, reasoning by analogy, we may conclude, from what is observed as the result of direct irritation of other mucous membranes, that the hurtful influences, to be mentioned further on, result not only in impairment of the gastric digestion, but also, certainly in most cases, in alterations in the structure of the walls of the stomach that can be demonstrated anatomically. It is obvious that irritants acting directly and energetically upon the gastric mucous membrane will excite inflammation in it, as they do in other mucous membranes under similar circumstances. Very hot or cold food, alcohol, spices, certain medicines, when their use is continued too long, and decaying food must be regarded as such. Similarly many of those kinds of food which are called difficult of digestion act as direct irritants upon the gastric mucous membrane by reason of their coarse texture. The more sensitive and irritable the inner surface of the stomach with which these irritants come into contact, the more apt is such action to take place. Thus, in young children, we often observe slight errors in diet give rise to gastritis, and among adults, those are most subject to gastric catarrh who are usually very careful in their choice of food, and have thus as it were weakened the gastric mucous membrane. With such persons a trifling indiscretion in eating or drinking, in which others might indulge without risk, may be sufficient to induce a decided gastritis. In this respect the infantile stomach is especially sensitive; it is known that the mother's milk alone, or at best those substitutes which resemble it most closely in composition, are assimilated by the child without disturbing the digestion and giving rise to gastric catarrh. Any change in this nourishment is usually felt unfavorably at once. It is known, further, that even the natural nourishment acts unfavorably upon the child if too much is taken at a time, and that the infantile organism is usually guarded against the bad effects of overfeeding only by the facility with which the excess of food is disposed of by vomiting.

A second cause of gastric inflammation is the too long retention of ingesta in the stomach. When excitation of the gastric mucous membrane, caused by the presence of food in this organ, is continued too long, a permanent irritation is the result, and gives rise to inflammation. The expulsion of the contents of the stomach is most probably brought about under normal circumstances by means of the collection of acid which takes place towards the end of digestion, and excites by reflex action the more violent muscular movements necessary thereto. Circumstances, therefore, which diminish the production of acid or prevent stronger contractions of the stomach, result in the retention of food in the organ, and consequently, indirectly, in gastritis.

We may take for granted a *decrease in the production of gastric juice* in all those cases where anæmia exists. This view is confirmed both by the clinical fact that *anæmic persons*, or *those who are badly nourished*, and particularly *small* and *delicate children*, are the ones who contract gastric catarrh most easily, and also by experiment; for Manassëin[1] showed by his physiological researches, carried out under the direction of Hoppe, that "in dogs, in which acute anæmia has been induced by bleeding, the normal relation between acid and pepsin is altered, and that, other things being equal, the addition of acid to the gastric juice in these animals has a greater influence than in healthy ones." Manassëin has also shown a similar relative lack of acid in animals suffering from fever. The long acknowledged fact, that *persons under the influence of fever* show a peculiar tendency to gastric catarrh, may therefore be explained most naturally by this lack of acid. If we consider, further, the observation of this author, that digestive mixtures made with the gastric juice of feverish animals quickly putrefy if not mixed with acid, and if we remember that food which has become putrid outside the organism is capable of exciting gastritis when introduced into the stomach, we shall have additional reason to regard the lack of acid in the gastric juice as a primary cause of catarrh of the stomach. In many of the above cases the occurrence of inflammation of the mucous membrane may be furthered by the diminution of the salivary secretion. The presence of saliva in the stomach, as was remarked in treating of the physiology of digestion, must be regarded as favoring the formation of gastric juice. Finally, in most of the conditions alluded to, besides the lack of acid, there is another factor to be taken into consideration—*weakness of the muscular structure of the stomach*, which delays the evacuation of its contents.

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[1] Virchow's Archiv. LV. pp. 451–452, and Centralbl. f. med. Wissen. 1871. p. 853.

The disturbing effect of this latter condition, involving as it does deficient energy in the movements of the stomach, and consequently incomplete expulsion of the chyme, must be evident; and in it is found the simplest explanation of the disposition to gastric catarrh observed in old persons, in certain cases of Bright's disease, and in affections of the heart and lungs. In these cases, apart from the alteration in the condition of the gastric vessels (vid. Chronic Gastric Catarrh), a slight serous infiltration of the muscular structure of the stomach takes place, accompanied by impairment of its functions.

Ordinarily the persistence of the causes of catarrh of the stomach just referred to is apt to give rise to the *chronic* form of gastritis; and yet, in the earlier stages of these pathological conditions this etiological influence may further the occurrence of *acute* gastritis by inducing a morbid sensitiveness to slight irritations which would not affect the healthy stomach unfavorably. Finally, in the severer forms of gastritis, inflammatory infiltration of the walls of the stomach by the fluid constituents of the blood, leading to a diminution of their vigor, is to be supposed. This would seem to explain the circumstance so frequently observed, that in acute gastric catarrh the food often remains undigested in the stomach for half a day or longer.

While the causes of acute gastric catarrh referred to thus far are connected with modifications in the character of the ingesta and in the transformations which they undergo, this is not the case with those generally known sources of the disease about to be mentioned. For reasons, as yet not clearly understood, many individuals are peculiarly prone to attacks of acute gastric catarrh after "catching cold," at certain periods of the year, especially in midsummer or during the prevalence of an epidemic of influenza. As regards any connection between erysipelas and gastritis, speculation is unnecessary. The latter was formerly supposed to be a complication of erysipelas, because a thickly-coated tongue, perverted taste, oppression in the region of the stomach, and vomiting are often associated with its appearance. The interdependence of the two affections need not be discussed until a series of post-mortem examinations shall have demonstrated that catarrh of the stomach is more frequently found in erysipelas than in other diseases terminating fatally after high fever.

Symptomatology.

The most constant sign of gastritis, compared with which the various other symptoms are secondary, is *vitiation of the appetite.* This is usually wanting, or only to be aroused by piquant, salt, or sour food, while an attempt to partake of the usual diet provokes disgust or nausea, the latter sensation being awakened in many patients at the mere sight of animal food. If the nausea goes on to *vomiting*, masses of mucus and food which may have been taken before, or even subsequent to, the onset of the attack, are rejected in a more or less altered condition. The matters vomited are occasionally mixed with bile, which must be regarded as an unfavorable complication, because this abnormal element in the contents of the stomach disturbs digestion still more. I cannot, however, regard as dangerous the occasional admixture of streaks of blood, which may at first sight arouse apprehension. In an extensive experience, gained by the use of the stomach-pump, I have learned how entirely immaterial this occurrence is, having found it in entirely healthy as well as in diseased stomachs.

The fact that *the ingesta may remain an abnormally long time in the diseased stomach* is beyond a doubt, and was confirmed especially by Beaumont's[1] interesting investigations, in which it was found that in feverish conditions, overloading of the stomach, etc., the food remained undigested in the patient's stomach for forty-eight hours and more. The principal cause of this is to be found in the imperfect gastric muscular movements alluded to, and a secondary cause in the fact observed at the same time, namely, that the gastric juice was no longer secreted. In consequence, the food is not digested normally, but rather *ferments*, gas and acid being formed in the stomach, occurrences which play an important part in connection with chronic catarrh of this organ, and which must be spoken of in greater detail under that head.

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[1] **Beaumont's Experiments and Observations on the Gastric Juice. 1838. p. 99.**

Together with the functional aberrations in the stomach itself, certain *changes occur in the action of the adjoining portions of the digestive tract, both above and below.* The former are shown by *heart-burn*, increase of the salivary secretion, perversion of taste, thirst, coating of the tongue, and *foul breath.* If the catarrh of the stomach extends downwards, *icterus*, *constipation*, or *diarrhœa*, with colic and the passage of offensive flatus, are observed.

Gastric catarrh exercises a very decided influence upon the *general nervous system.* A feeling of general malaise, bodily and mental depression, and severe frontal headache, are the usual accompaniments of acute gastritis, as most persons know from their own experience.

Senator, referring to a case of acute catarrh of the stomach, in which on the second day eructations, smelling of sulphuretted-hydrogen, dizziness, collapse, and frequent pulse were observed, and sulphuretted hydrogen was found in the urine, suggests that as in this, so in other cases, the nervous symptoms, so generally associated with abdominal affections, may be excited by the absorption of poisonous substances formed within the alimentary canal.

In addition, a dull, usually insignificant pain in the epigastric region is generally present, together with a feeling of weight and fulness in the abdomen.

In most cases of catarrh of the stomach the *bodily temperature* may be regarded as *increased*, though the exact amount of fever has only rarely been determined by direct measurement. The chilly sensations, alternating with transient feverishness, the marked general malaise, the hot head, the acceleration of the pulse, show unmistakably the existence of a febrile reaction, which may also be demonstrated by the thermometer.

Herpetic vesicles upon the lips, high-colored urine, with an abundant deposit, usually of the urates, appear in the course of the affection, and complete the picture, generally a very characteristic one, of acute gastritis.

In by far the greater number of cases the *course* of the affection is short and favorable. In some instances, though rarely, the febrile catarrh of the stomach is prolonged in its course and the feverish symptoms, together with the secondary nervous ones, become prominent; such cases have been separately classified under the name of *gastric fever*. This may render the diagnosis more difficult.

It frequently happens that when the stomach has once been affected relapses occur, and thus the inflammatory condition may become chronic, or the fever disappears and the disturbance of digestion persists. Simple, uncomplicated gastric catarrh *never* terminates *fatally*.

Quite different are the course and termination of those forms of acute catarrh of the stomach and intestines which I shall describe under separate heads, in part as variations of acute gastritis, in part as especial affections involving the stomach and intestines jointly—namely, cholera morbus and cholera infantum.

Diagnosis.

Uncomplicated gastric catarrh can not usually be mistaken for any other disease; the diagnosis becomes uncertain only when there is decided fever with marked general disturbance of the system, when in fact the case is one of "*gastric fever*," a term however better laid aside entirely. In such cases the question may arise whether the symptoms point to gastritis or to *typhoid fever*. It was in fact formerly held that the latter affection might be derived from the former; that gastric fever might run on to "nervous" fever. At the present time the differentiation of these essentially distinct morbid processes may be accomplished more certainly than in times past, since we possess in the thermometer a means of recognizing typhoid fever by its characteristic range of temperature. This aid to diagnosis can however only be made use of in fully developed typhoid, for in the lighter cases of this affection, the very ones which are to be discriminated from gastritis, the range of temperature varies decidedly, as Juergensen[1] has recently shown, from that presented by average cases. In cases, therefore, where the regular step-like rise in temperature in the initial stage, or the slight remissions of the fastigium of typhoid fever are absent, the diagnosis must be confirmed by other symptoms. Among these the chief is, that enlargement of the spleen which is observable, even in light cases of typhoid, at an early period, and is very rarely absent. The history also, which, in gastric catarrh, reveals some appreciable exciting cause of the affection, in typhoid fever points to infection, or at least to the simultaneous presence of other typhoid cases in the neighborhood of the patient. Furthermore, the appearance of herpetic vesicles is in favor of gastric catarrh, since these are quite common in this affection, while they occur only in a small percentage of typhoid cases. In many instances bronchitis and the appearance of roseola, which in light cases of typhoid may make its appearance even as early as the second day, will afford the assurance that typhoid and not acute gastritis is present.

In the early stage of *malarial fever* the symptoms may be so undeveloped and may seem so much like those of "gastric fever," that the discrimination of the two diseases will be almost impossible. Griesinger supposes, on this account, that many cases of intermittent, which recede without the completion of a typical paroxysm, are called gastric fever. Since, however, the spleen may already be somewhat swollen and painful, even in this equivocal period of intermittent, a careful examination of this organ, together with an attentive study of the patient's history, must be relied upon for establishing the differential diagnosis.

During the prodromal stage the other infectious diseases may, like intermittent, run a course closely resembling that of acute gastritis ; but the speedy appearance of their characteristic symptoms, the eruption of the exanthem, etc., will not leave the diagnosis long doubtful. Erysipelas, in particular, previous to the appearance of the reddening of the skin, resembles gastric catarrh closely.

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[1] Volkmann's Sammlung klin. Vorträge. No. 61.

Prognosis.

As already stated, uncomplicated catarrh of the stomach never ends fatally. The prognosis of the affection is in fact always *favorable*, so long as it runs an acute course. It is less so when the disease becomes chronic, or when the individual attacked is already debilitated or greatly advanced in years. The forms which have yet to be described as varieties of gastritis have, however, a decidedly less favorable prognosis, as will appear when we come to examine them individually.

Treatment.

In most cases of acute catarrh of the stomach a regulated diet suffices to keep the digestive disturbance within bounds. When the case is of a more serious nature, it is decidedly advisable to suspend the action of the stomach entirely for a while, causing the patient to fast several days or to receive nourishment by the rectum. If the case does not absolutely require this procedure, which is never pleasant for the patient, a bland, easily digestible diet may be prescribed, such as barley water, a couple of soft boiled eggs, beef tea (see Ulcer of the Stomach, p. 223), and the like. If this does not suffice, as in the severer cases of gastritis, treatment must be directed chiefly against the *exciting cause.*

This requires, in the first place, the removal of any portions of food improperly retained in the stomach, which have fermented, and whose presence keeps up irritation of the gastric mucous membrane. This may be effected by emesis or catharsis. The latter may be brought about by various agents—Carlsbad salts, rhubarb, or calcined magnesia, the latter being preferable, on account of its neutralizing effect upon the abnormal quantity of acid in the stomach. These and similar methods have, however, this disadvantage, that the undigested fermenting masses have to pass through the entire intestine before they are removed, and may irritate its mucous membrane also. On this account the better method would seem to be to remove the contents of the stomach by emesis. This may be accomplished by various emetics or by the use of the stomach pump. Among emetics, *apomorphia*, in doses of from a twelfth to a sixth of a grain, is to be preferred, because it may be used hypodermically, and also because it does not appear to exercise any injurious effect upon the mucous membrane of the stomach, while, on the other hand, tartar emetic, the chief of the emetics usually taken by the mouth, has recently been shown by Hermann and his pupils[1] to act by directly affecting the walls of the stomach, even when administered by injection into the veins. Since we also know that tartar emetic excites inflammation of the mucous membrane of the intestinal tract, the danger which lies in the use of this drug in gastritis is evident. But, even aside from the local effect of the remedy, the introduction of an emetic into the inflamed stomach is to be avoided, because during the act of vomiting the affected organ is compressed, and muscular movements, although slight ones, are excited in it. For this reason, the use of the stomach pump is in every way preferable to the administration of emetics. Further on, when considering the therapeutics of enlargement of the stomach, where the use of the pump plays so prominent a part, reference will be made more particularly to the various methods of cleansing or emptying the stomach, and I shall now only remark that in acute gastric catarrh, where the indication is for the removal of the masses of food remaining in the stomach, their simple evacuation is not sufficient; the stomach pump alone fulfils the requirements in these cases. When the contents of the stomach have been emptied by the pump, the mucous membrane of the organ may be advantageously cleansed by the introduction of a solution of soda (one or two teaspoonfuls to a quart of water) or a natural mineral water, such as Vichy or Ems water. This procedure is intended in part to neutralize the remaining acid by means of the carbonate of soda, and in part to further the process of digestion by means of the chloride of sodium contained in these waters.

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[1] Pflüger's Archiv. 1872. p. 280. Hermann's Toxikologie. 1874. p. 222.

After the stomach has thus been emptied of its irritating contents, the patient should be recommended to exercise for a time considerable caution in the matter of eating and drinking. This is most easily followed out by carefully adhering to the light diet above mentioned. In the case of anæmic or convalescent patients, the use of dilute *hydrochloric* acid is indicated in doses of from six to eight drops in a wine-glass of water, to be taken two hours after meals. It is just in this class of patients that lack of acid is a principal cause of acute gastritis, and a relapse of the disease may occur in consequence of the mucous membrane of the stomach, rendered sensitive by inflammation, being affected by the defective quality of the gastric juice.

In the case of gastric catarrh, brought on by catching cold, experience indicates *diaphoresis* as the proper treatment. Sweating may be most effectually brought about by hot baths (108° F.) with subsequent packing in a large blanket, in which the patient must remain for one or two hours. Nothing more is gained by this procedure than an energetic diversion of the blood from the internal organs, and in particular from the stomach to the skin, the blood-vessels of which seem to be dilated to their fullest extent. We must not, however, though this is commonly done, look upon diaphoresis as a remedy, the employment of which after catching cold is required by the causal indication. Diaphoresis is useful in gastritis resulting from the impression of cold only because the affection is here due, not as usual to the harmful effect of ingesta, but to sudden cooling of the blood (Rosenthal) and collateral hyperæmia of the walls of the stomach. To those who subscribe to this opinion it is hardly necessary to add, that after diaphoresis the temperature of the patient's surface must be reduced very gradually.

Should particular symptoms be especially prominent, the use of specific and appropriate remedies may be necessary in addition to those already mentioned. Thus, excessive vomiting or severe pain in the epigastrium calls for pellets of ice or the external application of ice-bladders, the exhibition of narcotics (for instance, a grain and a half of sulphate of morphia in five drachms of cherry-laurel water: dose from fifteen to twenty drops), and also the use of mustard plasters upon the epigastrium. Annoying acid eructation and heart-burn may render the administration of magnesia or some other antacid necessary. It must not be forgotten, however, that too large quantities of these remedies may produce alkalescence or at least neutralization of the gastric contents, and thereby only promote fermentation. Among the various antacids the carbonates of the alkalies, particularly the bicarbonate of soda, are to be preferred, because their carbonic acid, set free by the acids contained in the stomach, carries with it in its escape by eructation the various gases collected there, and thus prevents too great distention of the viscus or the absorption of deleterious gases, especially sulphuretted hydrogen. The relief which in certain cases patients experience, after the eructation which follows the ingestion of these salts, is thus explained.

Sporadic Cholera, Cholera Morbus (Cholera Infantum, Gastro-enteritis Infantum).

Certain forms of acute gastric catarrh with simultaneous inflammation of the intestine run a very peculiar course. The severity of the accompanying symptoms, at times apparently threatening life, resemble so closely those observed in Asiatic cholera that it is impossible to distinguish one disease from the other, unless regard is had to the absence or prevalence of epidemic cholera at the time.

Suddenly, or after faintness, nausea, and diarrhœa have existed for a longer or shorter time, the individual is attacked, usually in the night, by a feeling of anxiety, oppression in the epigastrium, and persistent vomiting, at first of food recently taken, later of watery, yellowish matter. At the same time there is continuous diarrhœa, one watery, slimy stool is succeeded by another, the passages in rare cases assuming even the well-known rice-water appearance. The choleraic picture is completed by insatiable thirst, the small pulse, hollowness of the pale or livid countenance, loss of the voice, cramps in the calves of the legs, ice-cold skin, and unmistakable general collapse.

It is not long before the question is settled as to the favorable or unfavorable termination of the case. The latter result occurs, as experience shows, most frequently among children and old people. Patients in the prime of life occasionally succumb, however, especially if the constitution has been previously undermined by enfeebling influences. For instance, some years ago, at a time when epidemic cholera was not prevalent, I remember to have observed a French prisoner of war succumb rapidly under an attack of simple sporadic cholera. In such cases the vomiting and diarrhœa cease, meteorism appears with weak retching, reptilian coldness of the extremities, cyanosis, pulselessness, and finally arrest of the heart. Should the attack take a favorable turn, as it generally does, the threatening symptoms shortly abate. The vomiting ceases, the diarrhœic stools become infrequent, the pulse fuller, the skin warmer, sleep supervenes, and the patient is convalescent. Yet for some days after the attack, irritability of the stomach, pain in the epigastrium, and slight diarrhœa persist.

The severer forms of acute gastric catarrh, usually accompanied by catarrh of the intestine as well, occur more frequently among *children* than among adults. They display an aspect similar to that just depicted. As a result of the constantly repeated vomiting, and the frequent, thin, watery, frothy or slimy, sour-smelling evacuations, the child weakens, the fontatanelle sinks, severe thirst is unmistakably expressed in the features, the urine is scanty, the movements are at first restless, the cries weak, the skin flabby, the extremities cool, while the body is often burning hot to the touch; the abdomen is tender on pressure, sometimes retracted, sometimes protuberant.

Amid these symptoms collapse may suddenly supervene: the child lies exhausted, with half-closed sunken eyes, failing pulse, the skin covered with clammy sweat, sometimes in quiet sopor, occasionally in convulsions and dyspnœa; or, with the development of localized pneumonias from the entrance of foreign bodies into the bronchi, the shocking scene terminates rapidly, perhaps even in a few hours, in death. In favorable cases the disease assumes a more chronic and less grave character, or often after a short time enters distinctly into the convalescent stage, which, however, may easily be disturbed by relapses.

Whatever may be the *cause* of these severe forms of the disease, it must be admitted that they are *peculiarly frequent, and make their appearance usually as little epidemics during midsummer and early autumn.* Certain general influences also are prevalent at this period, which favor the production of diseases of the stomach and intestines. Precisely what these are it is, unfortunately, in the present state of our knowledge, impossible to say.

The opportunity offered at these seasons for indulgence in unripe fruit is usually looked upon as a circumstance connected with the outbreak of the disease. We cannot, however, attach much importance to it, for diarrhœas begin early in the summer before fruit-eating has commenced, and moreover young children are most easily affected by cholera morbus.

That view which regards the rapid decay and putrefaction of food, particularly of that used by infants, which occur at this season, as one of the chief causes of severe gastric and intestinal catarrh, appears to be better supported. For, aside from those infantile diarrhœas which are often clearly the result of the use of sour milk, etc., cases have been known in which the eating of spoiled meat has undoubtedly caused choleraic intestinal catarrh.

In other cases, again, the affection may be induced by *nervous disturbances.* Thus Canstatt speaks of a lady, who, for sixteen years after an attack of intermittent fever, was subject to diarrhœa and vomiting which followed invariably upon any mental agitation. In addition, this patient suffered annually from an attack of cholera morbus, preceded by premonitory feverish symptoms.

It must therefore be admitted, in the light of the facts as at present known, *that the nerves of the gastric and intestinal parietes become irritated to a high degree, sometimes by the action of certain excitants, but more particularly by that of fermenting food, thus giving rise to excessive peristaltic action of the intestine and vomiting.*

The more sensitive the nervous system is to irritation in a given case, the more easily will this occur, which explains the frequency and obstinacy of cholera morbus in young children, as well as those instances in which the occurrence of cholera is undoubtedly connected with nervous influences. The correlative inference, that the nerves of the intestine react with peculiar intensity at certain seasons of the year, perhaps under the influence of miasmata generated just at these periods, must be disregarded for the present, although what has been said above gives certain positive grounds of support for such an hypothesis.

Post-mortem examinations of individuals dying of sporadic cholera fail occasionally to show any abnormal appearances in the mucous membrane of the intestinal tract, even when the most severe symptoms have been present during life. In such cases it is to be supposed, either that the hyperæmia connected with the gastro-enteritis has disappeared after death, because the inflammation was in the first stage of development, or, what is at least as plausible, that intense irritation of the intestinal nerves has occurred without resulting in essential structural changes in the walls of the alimentary tract. Still, in other cases marked alterations are observed: in particular, diffuse hyperæmia of the mucous membrane—in fact, the entire series of post-mortem appearances observed in Asiatic cholera, the huckleberry color of the blood, the sticky condition of the serous membranes, etc., may be present, as in the fatal case of sporadic cholera in an adult, mentioned above.

It is evident, from what has just been said, that the *diagnosis* between Asiatic and sporadic cholera can not be made even on the post-mortem table. The same holds good, as already intimated, in regard to the clinical diagnosis, when the disease occurs during the prevalence of Asiatic cholera. Under such circumstances, the mildness or malignity of the epidemic, the infection of different individuals from one and the same source, the number of cases existing at a given time, all point to one affection or the other. As a matter of course, in cases where the differential diagnosis between sporadic and Asiatic cholera comes into question, prudence demands that doubtful cases should be looked upon as cases of Asiatic cholera, and that the remedies appropriate to this disease should be promptly employed.

In by far the greater number of *adults* attacked by sporadic cholera, the *prognosis* is *decidedly favorable*, and this in spite of the apparently threatening symptoms. It may be accepted, in fact, that unless the patient is already in delicate health the attack rarely proves fatal in these cases. On the other hand, sporadic cholera is *especially fatal* among children, of whom a large number die of acute catarrh of the stomach and intestine, as is shown by the following table, taken from the post-mortem record of the Anatomico-pathological Institute in Erlangen.

Of one hundred and eighty-six children under one year of age dying during the period 1865–1874, forty-four succumbed to gastro-enteritis. The details[1] are as follows:

In 1865,	of 22	deaths,	there occurred	from gastro-enteritis	1
1866	“ 21	“	“	“	3
1867	“ 20	“	“	“	6
1868	“ 17	“	“	“	3
1869	“ 20	“	“	“	3
1870	“ 15	“	“	“	1
1871	“ 16	“	“	“	1
1872	“ 13	“	“	“	3
1873	“ 27	“	“	“	2
1874	“ 15	“	“	“	2

Total from 1865 to 1874—Deaths one hundred and eighty-six: of which twenty-five were due to gastro-enteritis; giving a percentage of 13.4.

Treatment.

In acute, violent, gastric and intestinal catarrh no remedy is so effectual as *opium*, which may be administered in the form of powder, in the dose of a third of a grain, or as laudanum in the dose of ten drops, repeated hourly in severe cases. Opium should be given to children as well as to adults when the vomiting and diarrhœa are excessive. The dose, of course, should be adjusted to the age of the child; on an average, perhaps, two drops of laudanum may be given through the day; as, for instance, a mixture may be made of five drops to three ounces of distilled water, or decoction of salep, of which a teaspoonful may be given every hour. Instead of this method of administration, the opium may be introduced, in the case of infants, in the form of enemata, composed of six and a half drachms of water, fifteen grains of starch, and one drop of laudanum.

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[1] The following cases only have been included: 1. Those in which the clinical diagnosis of gastro-enteritis was made, and where undoubted signs of the affection (gastritis, enteritis, with softening of the stomach) were found post-mostem. 2. Where, as in a few (four) cases, the post-mortem signs, while negative, did not contradict the clinical diagnosis, so far as the stomach and intestines were concerned (cases in which tuberculosis occurred as a complication were carefully excluded). 3. In addition those cases are included in which death having occurred without the establishment of a diagnosis, undoubted signs of gastro-enteritis were observed post-mortem.

While the restraining influence of opium upon vomiting and diarrhœa is very decided, that of the various other remedies recommended in these conditions is so to a less degree. They may, however, be employed in milder cases, where they will ordinarily be sufficient to check the discharges. I shall only allude briefly to the principal remedies. Chief among them, ranking next to opium, is *calomel*, which, administered to infants in the dose of a sixth of a grain every two hours, gives very good results. In the present state of our knowledge regarding the action of calomel, I am inclined to look upon its operation in these cases merely as that of a remedy which, while emptying the intestine and withal removing the irritating and frequently fermenting matters contained in it, seems to agree very well with the infantile organism. Next to calomel, *nitrate of silver* is most generally useful for the purpose of checking excessive diarrhœa. This remedy is most appropriate in the chronic stage of the disease, and may be given by the stomach according to the following formula: nitrate of silver, three-quarters of a grain: water, one and a half ounces: a teaspoonful every one or two hours. Nitrate of silver may also be administered in the form of enemata, three-quarters of a grain to three ounces of water. *Hydrochloric acid* has fewer advocates; it may be administered in doses of from seven to sixty minims, in six and a half ounces of water, according to the age of the patient. The effect of the acid depends, beyond doubt, upon its influence in assisting digestion, whereby the transformation of the food is hastened, its further decomposition checked, and the irritation of the intestinal mucous membrane diminished. The administration of *creosote* and *benzine* tends still more to prevent abnormal changes and decomposition in the contents of the stomach and intestine. The latter has been recommended by Naunyn as preventive of abnormal fermentation in the stomach. I have myself frequently used this remedy in cholera infantum, in the Jena Polyklinik, but without marked success. A further trial of it, however, might be advisable in cases like those under consideration. It may be administered to the adult in doses of twenty drops, in children to the amount of a few drops, twice daily.

The cure may be aided by the administration of pellets of ice, by mustard plasters applied to the abdomen, and the like. Should the icy coldness of the extremities increase, warm fomentations may be resorted to, or, still better, *hot baths.* By means of the latter, loss of body-heat may be decidedly diminished, and possibly collapse at least postponed. The baths should be followed by hot packing.

In order to strengthen the enfeebled action of the heart, *stimulants* are necessary, and their use cannot be long delayed in the treatment of choleraic attacks. Subcutaneous injections of ether to the amount of fifteen minims, repeated three or four times a day, camphor in doses of one and a half to four and a half grains, musk in doses of four and a half to eight, and the stronger wines are appropriate. Extreme caution in administering the latter to infants is not at all necessary; tokay may be given in teaspoonful doses even to children under one year of age.

The remedies just enumerated comprise of course only the principal means of treatment in the affection under consideration. In spite of their use, however, only transitory relief can be hoped for if the patient's diet be not at the same time strictly prescribed. Regulation of the *diet* constitutes, in fact, the principal method of treatment in sporadic cholera, and particularly in cholera infantum.

It must be made a rule that, at the beginning of a severe gastro-intestinal catarrh, the patient, particularly if a child, shall be *entirely* deprived of food, while the raging thirst is to be appeased, in the case of adults, with pellets of ice, and in infants with water which has been previously boiled. If in any individual case absolute fasting might seem to be not altogether without danger, the patient's nourishment should be confined to oatmeal gruel (oatmeal boiled down to a mucilage and strained), which may be given from a nursing-bottle. In cases where every other kind of food only increases the diarrhœa, this often answers surprisingly well. When necessary, red wine and tincture of opium may be added, so that medicine may be administered at the same time that food is given. Adults are to be confined to a diet of soups alone.

After the acute severity of the attack has passed, the greatest caution must be used in returning to the ordinary diet. Food must at first be given only in the smallest quantities and at considerable intervals.

Beef essence is most suitable for adults, together with soft-boiled eggs, broth, and similar easily digestible foods which will be alluded to more particularly when we come to consider the diet in chronic gastric catarrh.

Mother's milk is the only nourishment appropriate to the infant organism. Where this cannot be obtained, Liebig's "food" or Nestle's "lacteous farina" are alone to be recommended. The latter is especially commendable, because the physiological relations of the infantile digestive organs, particularly the lack of notable salivary and pancreatic secretion, are taken into account in its fabrication, the starch contained in it having at least been transformed into dextrin. Even in the case of this substitute for mother's milk, I cannot admit that the ideal nourishment in acute inflammatory conditions of the infantile gastro-intestinal tract has been attained. I am not able, however, to suggest anything better at present, as my experiments in the production of a more perfect artificial food are not yet completed.

To give *meat* to infants less than a year old, whether in the shape of scraped raw meat, or as beef tea—which latter is in every way preferable to the former, as being more digestible—I do not think advisable, since its clinical composition is too widely different from that most appropriate to the infantile digestive organs. The same may be said of eggs, pap made of dried rusk, meat broths, and all those "nourishing" foods which, with the best intentions, but without consideration of the limits of the infantile digestive powers, are forced upon the child suffering from gastro-enteritis, occasionally with the gravest consequences.

Gastritis Phlegmonosa.

Interstitial Purulent Inflammation of the Walls of the Stomach.

Syn: Gastritis submucosa, G. purulenta. Infiltration purulente des parois de l'estomac (*Reynaud*). Linitis suppurativa (*Brinton*).

In accordance with the usage which includes in the term "phlegmon" any inflammation of the connective tissue tending to suppuration, we designate by *gastritis phlegmonosa* an inflammation of the walls of the stomach, having its seat usually in the submucous layer, but which may also involve the intermuscular and subserous connective tissues. This form of inflammation is very rare, and the assemblage of symptoms which it presents is as yet by no means completely recorded.

The oldest observations on record date back to the latter half of the seventeenth and the beginning of the eighteenth century, and are those of P. Borel, 1656,[1] Sand, 1695,[2] Vorwaltner,[3] and Bonet.[4]

These authors allude to abscesses in the walls of the stomach, occasionally of considerable size (as large as a goose-egg in Bonet's case). In the first book of Lieutaud's "Historia Anatomico-Medica" there may even be found an entire chapter devoted to the "abscessus ventriculi," with the citation of seven observations by Riolanus, Bauhinus, and others. Andral and Cruveilhier[5] appear to have first observed a more diffuse purulent infiltration of the gastric walls. In their case the mucous membrane of the stomach was raised up from the muscular layer over its entire surface by a stratum of purulent matter, while it was itself at the same time so slightly affected that, as Cruveilhier remarks, only a fortunate accident, and the observation that the walls of the stomach were unusually thick, led him to make incisions into it, and thus to discover the separation.

In Germany, to Rokitansky, and still more to Dittrich,[6] belongs the credit of increasing our knowledge of this rare disease. The latter has himself observed several cases, and was the first to draw attention to the fact that gastritis phlegmonosa might terminate in entire recovery.

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[1] Opera, 1656.

[2] Diss. de raro ventriculi abscessu. Regiomont. 1701.

[3] Eph. Nat. cur. Dec. III. Obs. 142.

[4] Sepulchret. anatom. Lib. III.

[5] *Raynaud*, l. c., p. 526.

[6] See the Dissertations of his pupils, *Brand*, 1851, and *Clauss*, 1857.

In England, Habershon and Brinton, among others, have devoted their attention to the subject. Finally, the French physicians especially have been active of late in this direction, the treatise of Raynaud,[1] as well as Auvray's[2] monograph, which includes the observations of others, together with cases of his own, detailed with considerable completeness, deserving particular mention.

Pathological Anatomy.

The affection of the stomach under consideration occurs in two forms: as a circumscribed suppuration, abscess of the stomach; and as a diffuse, purulent infiltration of the parietes of this organ. The latter may be increased even to six times their normal thickness, while the abscesses may develop to the size of the fist.

Abscesses of the stomach are sometimes single, at other times multiple; their seat is usually in the submucous layer, although the suppuration may affect the subserous layer also, and the muscular stratum may take on fatty degeneration ("infiltration granulo-graisseuse," Auvray), and show interstitial proliferation. The mucous as well as the serous layer is usually intact, or at most only erythematous; only in rare cases is it so eroded that the contents of the abscess may escape into the cavity of the stomach or peritoneum. When the abscess has opened inwards, it may cicatrize, and under certain circumstances may bring about stenosis of the lumen of the stomach, or the abscess, after being opened, may be transformed into a chronic gastric ulcer under the influence of the acids of the stomach.

Cases of *diffuse purulent infiltration of the parietes of the stomach* have been observed less frequently and less exactly.

In this variety the *mucous membrane* of the stomach seems erythematous and swollen,[3] for the most part riddled like a sieve with holes varying in size from the head of a pin to a bean, and still larger; the smaller round, the larger irregularly eroded.

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[1] Gaz. hebdom. 1861. p. 511. et seq.

[2] Thèse. Paris. 1866.

[3] The cause of the swelling was found by *Hayem* (Obs. Morel) to lie in a purulent infiltration, demonstrated by the microscope, extending around and between the gastric glands.

The borders of these lesions of the mucous membrane are undermined, so that a probe may be pushed along under them, between the mucous and the submucous layers, for a considerable distance. On compressing the walls of the stomach, pus wells out from the openings in the mucous layer as from a sponge. In other cases (Tüngel, Asverus) the surface of the mucous membrane is intact. On cutting into it, the purulent collection, which is situated in the *submucous tissue*, is reached. The latter appears uniformly yellow, greatly thickened, and boggy, for the pus distends its meshes.

The condition of the *muscular layer* varied greatly in the different cases thus far examined ; it was sometimes unchanged, at other times infiltrated with serum or pus, the individual layers of muscular fibres being separated from each other by strata of pus. The infiltration is at times so extensive that the various tissues can no longer be distinguished apart. Occasionally the muscular layer may be destroyed and disappear entirely in the purulent accumulation, so that the latter may seem to be bounded outwardly by the serous layer and inwardly by the mucous membrane (Heyfelder).

Finally, the *serous layer*, like the muscular, may remain entirely unaffected, according to the seat and extent of the phlegmonous inflammation. In other cases it may take on inflammation, from the mildest to the severest grades, accompanied by the accumulation of dirty-gray, thick masses of exudation upon the surface of the stomach, which may glue it to the liver, intestines, etc. Upon cutting into the serous layer, the subserosa also is observed, in the more serious cases, to be infiltrated with serum or pus.

Notable as these anatomico-pathological changes are, yet, according to Dittrich, comparative reparation of the process is possible. The proof of this is in existence in the shape of two well-preserved preparations in our pathological collection which have come down from Dittrich's time.

In one of these a decided stricture of the pylorus has been developed as a result of proliferation of the connective tissue. "The mucous membrane at this point appears unchanged, drawn together in thick folds around the valve, while, without and below this, a partly suppurative, partly contractive process has taken place, which shows itself by the formation of a firm cicatricial tissue, with fistulous canals opening into the cavity of the stomach at one point. As an immediate result of this stricture of the pylorus, we find decided thickening of the mucous membrane in general, even that of the cardiac end."

The circumstance that the undermined mucous membrane does not perish from necrosis, but preserves its normal appearance, appears to indicate that the portions of tissue thus preserved are nourished from the contiguous parts. Later, the mucous membrane, riddled by fistulæ, may become thinned or may entirely disappear, through want of nourishment.

Etiology.

Among the thirty-one cases which have been observed and accurately described[1] up to the present time, twenty-six have been males and five females, so that the affection would seem to show a decided preference for the male sex, if the small number of cases does not mislead. That the latter is the case would seem most likely, since as yet we know of no especial cause for the occurrence of phlegmonous gastritis. In a number of cases the origin of the affection has been attributed to excessive indulgence in spirituous liquors; in a few to some wound occurring in the region of the stomach; in others, again, to some error of diet (in Stewart's case to excessive overloading of the stomach). These causes, among others, are cited as occasioning this severe affection of the stomach, although they may be present in a thousand instances without once giving rise to purulent gastritis.

We exclude here those examples of phlegmonous gastritis which occur during the course of the *severer infectious diseases*, particularly in *puerperal fever*. Here, at least, the original cause of those abscesses which appear in the walls of the stomach, as well as in other organs, has been settled, although tangible evidence as to the details of their formation is occasionally sought in vain. As in puerperal fever, where Dittrich observed the "frequent"[1] formation of these metastatic abscesses, so also in typhoid fever and variola these are observed, though not so often.

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[1] Twenty-two collected by Auvray; and, in addition, Vorwaltner's case, Dittrich's cases (one reported by Clauss, two by Brand), Wallmann's case, and, published since 1866, Stewart's case (Canstatt's Jahrbücher, 1868. p. 124), Loomis' (1870. p. 156), that of Malmsten and Key (1871. p. 148), and Krause's (Diss. Berol. 1872).

Two distinct forms of purulent gastritis must thus be acknowledged—the primary *idiopathic*, and the secondary *metastatic.* In the above description of phlegmonous gastritis the former variety alone has been considered, since the latter is almost invariably obscured during life by the symptoms of the main disorder, and is observed only as an accessory, together with various other metastatic products of inflammation, upon post-mortem examination.

As regards *age*, those who died of gastritis phlegmonosa were almost without exception in middle life.

Symptomatology.

It would be impossible to give a distinctly-outlined description of the disease under consideration, such as would apply to every case, because as yet it has been too unfrequently observed. Such a description is the more difficult because peritonitis, to a more or less marked degree, occurs in all cases in connection with the diffuse phlegmonous inflammation of the walls of the stomach. It is therefore difficult to tell in any given case which symptom belongs to the peritoneal inflammation and which is to be ascribed to purulent gastritis.

As regards this point, the diagnosis is clearer in those cases where the purulent inflammation of the gastric parietes is limited in the form of abscesses, since cases of this kind occurred without the accompaniment of peritonitis. Little is to be gained from the earlier literature of this variety in relation to the symptomatology of abscess of the stomach, because the observations are too briefly recorded.[2]

The patient usually suffers from pain in the epigastrium and vomiting, with slow fever; occasionally there is some difficulty in respiration. In two cases (Lieutaud, l. c., No. 86, and Sand) a tumor, which in one case was as large as the fist, and not particularly painful upon pressure, could be felt in the epigastrium.

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[1] In an epidemic in Prague in the year 1847; see *Brand*, Diss. Erl. 1851. p. 28.

[2] Sand's case (1695) is to be excepted, a full description of the disease being given.

In the latter (Sand's patient) the abscess appears to have ruptured during life, discharging a large quantity of dark-colored matter. The rupture of the abscess in Callow's case (1824) is established beyond doubt. Here *twenty ounces of pure pus were vomited in the presence of the physician*, and some was also passed at stool. In the same case post-mortem examination showed more than seven pounds of pus in the abdomen, derived from an enormous abscess of the stomach, which had brought about the fatal result by perforation, without having previously caused pain or any other symptom.

Abscess of the stomach may follow an *acute* or *chronic* course. While the earlier recorded cases took the latter form, recent literature affords several examples of acute gastric abscess.[1] The aspect of the disease in these cases was as follows. After loss of appetite with colic and irregularity of bowels, lasting several days, the disease itself set in with pain in the abdomen and vomiting. The pain was generally diffused, in spite of the absence of peritonitis ; it was particularly violent, however, only in the epigastrium, and was increased by pressure at this point. Together with these symptoms there was continued want of appetite and diarrhœa, the latter in one case only towards the end of life ; severe thirst, with dryness of the tongue, showed concomitant fever, which is also especially mentioned in one case. Finally, small and irregular pulse with delirium set in, and death followed with symptoms of general prostration and coma. The duration of the disease, from the beginning to the fatal result, was in one case eight days, in both the others about two and a half weeks.

In contrast to these circumscribed abscesses of the stomach, there have been observed, during the past forty years, from twelve to fifteen cases of *diffuse suppuration of the walls of the stomach*, in which death followed in a short time, after severe general symptoms accompanied by peritonitis.

If two cases are excluded, in which the process took a chronic course and the fatal result only occurred after some months, the duration of the disease may be said to vary from three to eighteen days, the average being nine days.

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[1] Cases by Habershon, 1847, with very scanty notes, by Dumesnil, 1861, and Auvray, 1866 ; the above description is founded on the last two.

Among the clinical histories upon which the description of diffuse purulent infiltration of the walls of the stomach has been founded, that obtained from Dittrich[1] is one of the best. I shall, therefore, recount this before going on to describe the general aspect of the disease. For when the small number of cases observed is taken into consideration, it may easily be understood that *one* well-recorded case is of more value than a general description.

G. S., a tanner, entirely convalescent for two weeks from pleuritis, felt perfectly well on the 20th of October, 1856, dined with a good appetite, and spent the day at work as usual. On returning home, towards evening, he experienced *severe chills, alternating with fever and accompanied by intense pain in the region of the stomach, heart, and left thorax*, so that he could not sleep that night.

On the morning of October 21st *repeated vomiting of bilious matters* took place, while the former symptoms continued. In the evening he presented the following symptoms: *His general appearance was that of a very sick person, his temperature was decidedly increased, his pulse one hundred and twelve, strong and full.* Nothing abnormal about the heart, some râles through the chest. *The abdomen was not meteoric, but soft, and not painful on pressure.* No annoyance from constipation of two days' duration. In addition, the patient complained of pain, varying in intensity, in the stomach and left side, nausea, eructation, and occasional vomiting. He was ordered: one-twelfth of a grain of acetate of morphia, four times during the night.

October 22d. Restless and sleepless during the past night; *vomiting occurred repeatedly*, but ceased in the morning; coughing and lividity; intensity of the pain continued the same evening; ordered eight cups, morphia, and an enema to relieve the constipation.

October 23d. Better night, some sleep, pain relieved by the cupping, *no more vomiting, less fever, some appetite.* General appearance better.

October 24th. *Decidedly worse during the night*, entire sleeplessness, continual pain, with occasional *delirium*, starting up and jumping out of bed. Temperature considerably higher, face flushed, and cheeks hollow; *pulse very rapid*, moderately strong and *soft. Intense thirst.* Some sleep through the day, with light delirium, under the influence of morphia. On waking, complained of severe pain in the region of the left nipple. Respiration short and laborious.

During the first part of the following night the patient was very restless, the delirium continued, and he repeatedly sprang out of bed. After midnight he became somewhat quieter. Toward morning *collapse* supervened, which, in spite of the use of stimulants, terminated fatally on the morning of October 25th.

Post-mortem examination revealed independent *idiopathic inflammation of the*

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[1] *Clauss's* Dissertation.

stomach, in the form of diffuse sero-purulent infiltration of the submucous layer, gradually extending over the other membranes (peritonitis, pleuritis). There was *general dissolution of the blood.*

It is very remarkable that, in this as in other cases of gastritis phlegmonosa, *the pain was not increased by pressure over the epigastrium*, as would be expected in so severe an acute inflammation of the entire parietes of the stomach. The most *constant symptom* in this affection is the *vomiting.* This was absent in only one of the cases recorded, and even then there was a disposition to emesis. *The vomited matters contained only bile and mucus—never, so far as known, pus.* If we may judge from the anatomico-pathological appearances, pus had without doubt percolated into the stomach through the sieve-like apertures in the mucous membrane, but was either digested there, or became so intimately mingled with the gastric contents as to escape detection by the naked eye.

Among the other symptoms, *delirium*, or at least *restlessness* and terror, almost always supervened. In one instance the latter was so intolerable as to drive the patient to suicide.

In the greater number of cases, meteorism, diarrhœa, severe thirst, and dry tongue, were observed. The pulse also was small and very rapid during the later periods of the disease, even when in some cases it had been full and strong at the beginning. It is difficult to say just how far these symptoms are to be referred to the concomitant peritonitis and consequent fever. Unfortunately, in regard to the latter, exact observations have been made in only a few (four) instances.[1]

The general aspect of the patient was that of a person evidently *very sick;* this appearance is in part to be attributed to the peritonitis accompanying the gastric affection, as well as to the *collapse* which in most cases preceded death.

The assemblage of symptoms offered by patients suffering from phlegmonous gastritis is, briefly, about as follows: In the midst of perfect health, or after a period of general malaise, the individual is seized with pain in the stomach and vomiting, accompanied by thirst, dry tongue, small, frequent irregular pulse, meteorism, and diarrhœa; subsequently delirium and prostration ensue, and finally death.

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[1] In Krause's case the temperature varied between 102.2° and 104° F.

Diagnosis.

After what has just been said, it need hardly be added that the diagnosis of diffuse purulent infiltration of the parietes of the stomach can *not* be established, in the present state of our knowledge, during life, since the symptoms observed may all be attributed, not unnaturally, to the accompanying peritonitis. In those cases where the gastric symptoms above mentioned are much more prominent than the general symptoms of peritonitis and where other diseases of the stomach giving rise to peritonitis may reasonably be excluded, the possibility of phlegmonous gastritis might at least be taken into consideration. The diagnosis of *acute gastric abscess* is not less difficult, because, as is evident from the description above given, the symptoms in this affection correspond to those of diffuse purulent infiltration. It is only when the abscess can be felt as a circumscribed tumor in the epigastric region, and when it proceeds rapidly to rupture into the stomach, with vomiting of pus, that the diagnosis can be thought of. Since these last two diagnostic signs have been actually present in some cases of *chronic* abscess of the stomach, a distinct tumor having been felt in the epigastrium, and in one case (Callow's) pus having been discharged in great quantity, *the possibility of diagnosticating chronic abscess of the stomach must be conceded.* That the various other symptoms observed in chronic abscess of the stomach—vomiting, pain in the epigastrium, slow fever, difficult respiration—are valueless as aids in forming the diagnosis, is self-evident; the more perfectly so, since just in that striking case, where the purulent contents of the abscess were vomited, scarcely a symptom was manifest up to the moment when rupture into the cavity of the stomach took place, a day and a half before death occurred.

Prognosis.

The prognosis in this affection is grave. The disease cannot, however, be regarded as absolutely fatal, since Dittrich's preparations show that a comparative cure may take place (vide supra). On the other hand, Callow's case, to which allusion has just been made, shows that even enormous abscesses may develop gradually, almost without a symptom,[1] and may yet suddenly terminate fatally.

Treatment.

It is evident that, so far as direct remedies in purulent gastritis are concerned, nothing can be offered; since, as yet, it has not been possible to diagnosticate the disease during life. The individual symptoms, particularly the peritonitis, are really the only points against which therapeutic measures can be directed.

In addition to diffuse inflammation of the stomach, there are other varieties, which, like phlegmonous gastritis, may, when more extensively developed, attack the deeper layers of the gastric structure; but these differ from the phlegmonous form in the circumstance that they originate in the mucous membrane, and only involve the deeper layers secondarily. Such are *diphtheritic gastritis, and those changes in the structure of the stomach which result from the action of corrosive substances.*

The first—*diphtheritic gastritis*—is a very rare affection. It sometimes arises simply from extension of the diphtheritic inflammation of the pharynx and œsophagus to the mucous membrane of the stomach; at other times as a sequela of typhoid fever, pyæmia, particularly puerperal fever, or of the acute exanthemata, especially small-pox. Finally, diphtheritic gastritis occasionally occurs in persons who are very much debilitated, and in infants. It makes its appearance "in the form of yellow, yellowish-white, or brownish eschars of the mucous membrane, each surrounded by an areola of injection, and the removal of these shows decided losses of substance, at the base of which the submucous connective tissue may be seen laid bare, reddened, injected, or reduced to a gangrenous, pulpy, brownish-black mass" (Rokitansky). Should any vessels be destroyed by this process, hemorrhage may occur, which in the case of young children may terminate fatally.

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[1] The patient alluded to continued his occupation as cook up to the very day on which the abscess broke, and only complained to his comrades towards the last that his strength had diminished, and that he could not retain food in his stomach for any length of time.

This variety of gastric inflammation possesses no clinical significance, because the symptoms peculiar to it are masked by those of the primary affection; or, if the gastric symptoms are more prominent, they do not differ essentially from those of a simple gastric catarrh.

The symptoms caused by *erosion of the surface of the stomach by corrosive substances* are presented more frequently, and, from a clinical point of view, are more interesting. They vary according to the nature and concentration of the caustic, and constitute an essential feature in the general aspect of the disease, which varies according to the poison concerned. A systematic description will be found in another place. (See the volume on Poisons.)

Chronic Gastritis.

Chronic catarrh of the stomach is a very common disease, if we include under this designation those cases in which chronic hyperæmia and inflammation of the gastric mucous membrane occur in the course of other affections, as the immediate, and for the most part mechanical, consequence of the principal disease, or where, on the other hand, dilatation of the stomach supervenes upon an existing gastric catarrh. The latter (dilatation), whether originating in one way or in another, is an especial disease with symptoms peculiar to itself, and on that account must be dealt with under another head.

Setting aside these secondary forms of gastric catarrh, and excluding also dilatation of the stomach from the division of chronic gastritis, *chronic catarrh of the stomach occurs, at least* *according to my own experience, only very unfrequently, as an independent disease*, and I consider the custom which has lately become usual, of regarding persistent dyspepsia as chronic catarrh of the stomach, as a decided mistake in diagnosis. *Rather would I establish it as a rule that we should not be satisfied with the diagnosis of a primary chronic gastric catarrh until the various other chronic affections of the stomach giving rise to dyspepsia can be excluded with tolerable certainty.*

Pathological Anatomy.

In chronic gastric catarrh, as in other morbid processes affecting the walls of the stomach, the pyloric portion is attacked by preference. The mucous membrane is sometimes red or purplish red, sometimes pale, with traces of previous hyperæmia, usually in the form of dark patches, which are produced by the deposit of *pigment granules* in the interstitial tissue, especially in the delicate villi of the surface or in the cells of the glands themselves. This pigment is a product of the metamorphosis of the coloring matter of the blood deposited at these points by capillary hemorrhages, or by the extravasation of large quantities of red blood cells under the influence of severe and persistent inflammation. The inner surface of the stomach is often coated with a thick layer of tough gray or yellowish mucus. The *mucous membrane* also usually appears *thickened*, and the changes due to inflammation are present; that is, hypertrophy manifests itself in various places and in very different ways.

The proliferation of the *interstitial tissue* causes this to protrude between the gland tubules in the form of conical projections, under the pressure of which, in rare cases, the tubules may become atrophied. At other times the *gland* tubules themselves become the subjects of this new cell growth, and under these circumstances lengthen and bulge out, while the cells in their interior—especially the principal cells—swell and undergo partial fatty metamorphosis. Since, as a result of this proliferation of the mucous membrane generally, its area becomes more extensive than that of the subjacent muscular layer, the inner surface of the stomach is thrown into mammillary elevations, which are larger than those normally present during life, and on that account do not, like the latter, disappear after death upon the cessation of muscular contractility (état mamelonné).

The proliferation of the interstitial tissue in general brings about a thickening of the limiting membrane of the tubules, which on its part gives rise again to strangulation of the glandular portion, accumulation of the contents of the gland behind the obstructed point, and the formation of small *cysts* projecting upon the gastric mucous membrane.

In addition to this fatty metamorphosis in the glands, fatty degeneration of the connective tissue is also observed in a certain number of cases (W. Fox), and in connection with this a disintegration of the surface resembling "fatty erosion."

If the proliferation of the glandular exceeds that of the interstitial tissue, globular non-projecting tumors (adenomata) are formed.

In addition to the alterations in the mucous membrane, just described, chronic gastric catarrh gives rise to changes in the deeper structures of the stomach. The *submucous layer* becomes thickened and coarsely fibrous. In very marked cases, where the inflammation has affected this layer more particularly, its thickness is increased ten or twenty fold. Such were probably the cases which Brinton represented as a peculiar form of cirrhotic inflammation of the stomach.

When this hypertrophy of the submucosa is circumscribed, the overlying mucous membrane, which is usually atrophied at those points, projects in *polypoid forms* above the inner surface of the stomach ("gastritis polyposa").

Moreover, since the interstitial connective tissue between the muscular fibres increases in volume, and the *muscular* coat itself becomes hypertrophied, an appearance is presented similar to that very often observed in scirrhus ventriculi, namely, the tissue when exposed to view seems to be divided up into numerous compartments, caused by the interpenetration of the grayish-red muscular fibres by the white bands of proliferated intermuscular connective tissue. In connection with these anatomical changes in the walls of the stomach, stenosis of this organ commonly takes place at the same time, which, in case the pyloric region especially is affected, may hinder the passage of food, and give rise to consecutive dilatation.

Finally, the *serous layer* is also occasionally involved in the chronic inflammatory process, the subserous layer becomes adherent, the epithelium disappears to a greater or less extent, and the whole membrane appears thickened and rough.

Etiology.

It is evident that the same group of causes which occasion acute gastric catarrh may give rise to chronic inflammation of the stomach, when their action is continuous or recurs so frequently that the effect of one irritation is not entirely recovered from before that of the succeeding one is felt.

Among the predisposing causes *anæmia*, *chlorosis*, etc., as has been already intimated, are prominent, since, in individuals thus affected, the weakness of digestion which accompanies these conditions favors the occurrence of chronic gastric catarrh.

Among the irritants which affect the gastric mucous membrane directly, those which are in most common use, as certain drugs, hot spices, and the like, give rise most readily to chronic catarrh of the stomach. The habitual excessive use of *alcoholic liquors*, particularly the concentrated forms, as brandy, are more injurious in this way than other ingesta.

Besides these irritants introduced from without, others of an *internal* nature, dependent upon changes going on in the structure of the stomach itself, must be regarded as giving rise to chronic gastric catarrh. Thus the growth of tumors in the walls of the stomach, gastric ulcers, etc., occasion chronic inflammation in their vicinity, and a part of the appearances which are observed in the course of these affections of the stomach may be attributed to the complicating gastric catarrh. This will be discussed more fully under the appropriate head.

The occurrence of chronic gastritis is also favored by various morbid processes which bring about permanent alterations in the condition of the gastric vessels, or, as Cohnheim's remarkable researches upon inflammation warrant us in asserting, in the structure of the walls of these vessels. We must therefore for the present regard the occurrence of chronic gastric catarrh in patients suffering from Bright's disease, cancerous cachexia, tuberculosis, etc., as depending upon the fact that the physiological integrity of the vascular walls gradually suffers under the influence of that waste of albumen and tissue so prominent in these affections. In this way opportunity is offered for the occurrence of inflammation. A predisposition to disease once established by any one of the affections just mentioned, an organ, which like the stomach is constantly exposed to the action of external irritants, sometimes of a very coarse kind, is strongly prone to take on inflammatory action.

The fact that chronic catarrh of the stomach is a complication of those morbid conditions which are accompanied by persistent hyperæmia of this organ, may be explained in a similar manner. Such a condition is observed chiefly in *obstruction of the portal system*, as well as in various *affections of the liver*, particularly *cirrhosis*, and in *diseases of the portal vein itself*. Furthermore, *impediments in the system of the vena cava*, dependent upon *affections of the lungs and heart*, give rise to obstructions in the parietes of the stomach—in part directly, through the inferior œsophageal veins, in part indirectly, through the hepatic veins, the venæ centrales hepatis, and the trunk of the portal vein with its gastric branches. In all these cases, while the obstruction itself is not the immediate cause of inflammation, it seems at least to induce a predisposition to this condition. Thus irritants coming in contact with the mucous membrane of the stomach under these circumstances, though less severe than those frequently borne by it with impunity when in a normal condition, are sufficient to arouse inflammatory processes in the vessels and surrounding tissues. These favor serous infiltration of the muscular structure of the stomach with the resultant enfeebled gastric movements.

The more frequent occurrence of chronic gastric catarrh in the male sex doubtless depends upon the fact that men are more exposed than women to the causes which induce this affection. As regards the circumstance that aged persons are more easily affected than the young, this is accounted for by the weakened condition of the muscular structures, and also of the vascular system of the stomach,—in other words, by those causes which tend, as we have already mentioned, to favor the occurrence of gastric inflammation.

Symptomatology.

The chief symptom of chronic catarrh of the stomach is, as might be expected, difficulty of digestion—"*dyspepsia.*" Dyspepsia is a complex of symptoms which in a general sense may be said to be present in all gastric affections, because, after all, it is nothing more than the expression of functional disturbance on the part of the digestive apparatus, especially of the stomach itself. When it is considered that the first, if not the only requisite for perfect digestion is, that a sufficient quantity of active gastric juice shall be secreted, it may easily be understood that in no disease are the symptoms of dyspepsia more decided than in affections of the mucous membrane, this itself containing the source of the gastric juice. Catarrhal inflammation is of all the affections of the stomach the one best calculated to give rise to dyspepsia, because the disease usually affects considerable areas of the inner surface of this organ.

The diagnosis of dyspepsia presents no difficulties. It shows itself first in *diminished or perverted appetite*, there being at times a strong craving for piquant foods, at other times an aversion to all nourishment. For instance, almost as soon as the patient has begun to eat, he experiences a feeling of repletion, by which he must take warning to eat no more, if he does not desire to risk an increase of discomfort after the meal. In connection with this, there is frequently an *increased secretion of saliva*, probably excited by reflex action from the stomach, together with *perversion of taste*, most naturally to be accounted for by the concurrent catarrh of the mouth and pharynx.

Neither abnormal thirst nor alterations in the appearance of the tongue are to be regarded as constant symptoms. On the other hand, an *uncomfortable sense of weight or pain in the epigastrium*, a desire to gain relief by sighing, together with lassitude, a dull feeling in the head, etc., are all symptoms which usually accompany gastric catarrh.

Much more important than the symptoms just mentioned are the objective signs—the only positive indications of the presence of dyspepsia. I allude to *those variations from the normal standard in the composition and transformations of the con-*

tents of the stomach, which can be estimated by chemical investigations. Many patients suffer from acid eructation, that is, the rise into the mouth of fluids possessing an acid taste. This is to a certain degree characteristic of chronic gastric catarrh. Fenwick has shown, by examinations in a number of different cases, that, when persons who suffer *constantly* from acid eructations after eating are caused to vomit before breakfast, large quantities of tough mucus, mixed with the remains of food, are ejected; while in persons who only suffer from eructation to a less degree, or in whom vomiting is only occasionally induced, a small quantity of thin fluid is ejected. Since these examinations were made in persons who had not yet breakfasted, they have a decided value, for, according to my experience, the stomach of healthy persons is at this time entirely empty.

The inquiry as to the cause of this acid eructation is connected with the question as to the origin of the acid present in the contents of the stomach *vomited* in gastric catarrh. Examination of the *matters vomited* by patients suffering from this affection shows the presence of acids of the fatty series, particularly *acetic acid* and *butyric acid.*

Lactic acid, which is also found, cannot be regarded as an abnormal constituent, since it is present as a normal digestive product of the carbohydrates. The formation of lactic acid may probably be regarded as a normal *process of fermentation* going on in the healthy stomach, by which the starchy portions of the food are transformed, under the influence of some ferment introduced into the stomach from without. The saliva here plays only a subordinate part. Since, in addition, it is known that in gastric catarrh the food remains a longer time in the stomach, the thought naturally suggests itself that under these altered circumstances the action of the ferment is more persistent, and the normal fermentation proceeds to a further extent. More than the normal amount of lactic acid is also formed in the stomach, and this during the fermentative process goes on to the formation of *butyric acid*,[1] with the development of *carbonic acid* and *hydrogen.* This metamorphosis can, as is known, be brought about outside of the body, when putrefying albumen is allowed to act upon sugar, or when the salts of lactic acid are caused to ferment. Besides these two acids, *acetic acid*[2] is also formed from the carbohydrates in the diseased stomach.

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[1] $$\underset{\text{(Grape sugar.)}}{C_6H_{12}O_6} = \underset{\text{(Lactic acid.)}}{2C_3H_6O_3} = \begin{cases} C_4H_8O_2 & \text{Butyric acid.} \\ C_2\quad O_4 & \text{Carbonic acid.} \\ \quad H_4 & \text{Hydrogen.} \end{cases}$$

[2] $$\underset{\text{(Grape sugar.)}}{C_6H_{12}O_6} = \left\{\begin{array}{l} \left.\begin{array}{l} C_2H_6O \\ C_2H_6O \end{array}\right\} \text{Alcohol} + {}_4O = \begin{cases} C_2H_4O_2 & \text{Acetic acid.} \\ C_2H_4O_2 & \\ \quad H_4O_2 & \text{Water.} \end{cases} \\ C_2\quad O_4 \text{ Carbonic acid.} \end{array}\right.$$

Frerichs, to whose valuable labors upon the subject of digestion we owe the first explanation of these fermentative processes in the stomach, demonstrated *yeast fungus* in the matters vomited, while Graham and Schultzen showed the presence of alcohol, as a result of the transformation of carbohydrates. It thus appears beyond a doubt that, under certain circumstances, alcoholic fermentation may take place in this organ.[1]

We must, therefore, distinguish two different directions in which the transformation of the carbohydrates tends. On the one hand, we have *butyric acid fermentation*, in the course of which lactic acid, butyric acid, carbonic acid, and hydrogen appear as the principal products of change, while, most probably, other members of the fatty acid series (formic acid, acetic acid, caprylic acid, etc.) are found as secondary products. On the other hand, we have *alcoholic fermentation*, with carbonic acid and acetic acid as the principal products, glycerine, and succinic acid (Schultzen) as secondary products. These are beyond doubt formed by the development of alcohol in the stomach, just as alcohol and carbonic acid result, according to Pasteur's observations, from the fermentative transformation of sugar.

These fermentative processes are initiated, partly by ferments introduced into the stomach from without, which come into play during the transformation of the gluten into lactic acid, and during yeast fermentation, and partly, by the contact of the carbohydrates, particularly sugar, with albuminous substances which have begun to putrefy. Since, however, the gastric juice usually *hinders putrefaction;* since, further, it has been observed that yeast fungus may be swallowed by the ounce without injurious effect, the normal gastric juice evidently preventing the occurrence of fermentation as well as putrefaction to any extent, we must, if we would understand the anomalous transformations which occur in the stomach during digestion in cases of chronic gastritis, ask ourselves the question, *in what way the normal composition or action of the gastric juice is altered.*

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[1] Frerichs found, in addition, that "a part of the carbohydrates" in the matters vomited "were changed into a tough, stringy mass," and he ascertained by the post-mortem examination of a patient, who during life had ejected masses of this nature with great effort, that the walls of the stomach were coated with a layer of viscid matter from two to three lines in thickness. This transformation of sugar into a mucilaginous material like gum-arabic is also observed outside of the organism—in beet juice, for example—and is known by the name of "*viscous fermentation.*" This variety of fermentation forms the third kind of abnormal transformation which the carbohydrates may undergo in the stomach.

As yet it is impossible to decide this question with certainty in all cases, but the following explanation will, it is hoped, afford some aid in this direction.

As regards the two constituents of gastric juice, acid and pepsin, certain relative proportions are necessary to normal digestion. The amount of acid present in the stomach is of peculiar importance as regards the undisturbed fulfilment of the process. For experiment has shown that the smallest amount of the other constituent of gastric juice, pepsin, is sufficient to dissolve an unlimited quantity of fibrin, if a proper supply of acid is constantly kept up. An abnormal *diminution of the proportion of pepsin* in the gastric juice will not put a stop to digestion, but, as experience has demonstrated, will at most retard this process to a certain extent. A *want of acid*, on the other hand, will soon bring digestion to a stand-still.

While now, as we have seen above, there can be no doubt that in acute gastric catarrh deficiency of acid must in many cases be regarded as the cause of defective digestion, this by no means accounts for the dyspepsia of chronic gastric catarrh. On the contrary, the belief is widely prevalent that in this affection an excess of acid is present in the stomach, of which the acid eructation and vomiting of chronic gastritis is an undoubted symptom. Occasionally, as I have convinced myself, the contents of the stomach, though possessing a disagreeable acid odor, may be neutral in reaction. Aside from this, the decision of the question, as to excess or deficiency of acid in the stomach, depends upon the nature of the acid present. For since experiment has shown that hydrochloric acid possesses, in connection with pepsin, about ten times the digestive power of acetic acid, *it is evident that the gastric juice of a patient suffering from chronic gastric catarrh, which is apparently rich in acid, may, as regards its digestive power, be really poor in this respect, the acid present being merely acetic acid, derived from the decomposed carbohydrates of the food.* This theory is verified by clinical observation; for, if a portion of its contents be removed from the diseased stomach by means of the pump, it will be found that, although this may offer a strongly acid reaction, yet, upon neutralizing it slightly and adding pepsin, so far from its digestive power being increased, it is diminished.

At all events, after taking into account the various circumstances connected with dyspepsia, the physiological experiments which have been made, together with my clinical researches, and, finally, upon consideration of the beneficial effects of hydrochloric acid in chronic digestive derangements, it must be admitted, *that, in many cases of dyspepsia, a lack of acid is the cause of defective digestion.*

It is not indeed to be denied that the absolute quantity of acid present in the stomach in the dyspepsia of chronic gastric catarrh may be very considerable; only, as we have seen, the quality of this acid is such that it is not calculated to effect active digestion with pepsin. Were the hydrochloric acid itself abnormally increased in quantity, no evil could result unless this increase was excessive. The latter can hardly be imagined, however, since the limits within which the proportion of acid may vary without digestion suffering to any extent are pretty wide, and, besides this, the excessive increase of the acid present would cause a speedy expulsion of the contents of the stomach. It is quite as improbable that an abnormal increase in the amount of pepsin should interfere with the digestive process for any length of time; for a larger proportion of pepsin in the gastric juice would, on the contrary, accelerate digestion, and even if an excess were present, this would be reabsorbed without injurious effects.

On the other hand, an excessive accumulation in the stomach of the product of the action of pepsin and acid—the peptones—is probably a not infrequent cause of chronic dyspepsia, particularly in chronic catarrh of the stomach. We know that even twelve hours subsequent to a full meal of animal food, acid gastric juice, together with a large quantity of undigested meat, may be ejected, and we must in these cases conclude that the ingestion of too large a quantity of albuminous matters prevents the accomplishment of perfect digestion. This is still more apt to be the case when the muscular structures act inefficiently, which, if we may judge by pathological appearances, must be the case in chronic gastric catarrh. If we consider, further, the fact, which is noted daily, that in chronic inflammation of other organs resorption is retarded, and also that in chronic gastric catarrh decided changes must probably occur in the walls of the vessels, and that this, in connection with the thick layer of mucus which is spread out over the inner coat of the stomach, acts still further to diminish resorption, we can scarcely doubt that deficiency in this latter process is among the causes of dyspepsia in chronic gastric catarrh. The actual existence and etiological significance of this factor will be more particularly discussed in the section upon dilatation of the stomach. Abnormal retention of the products of digestion in the stomach, whether due to one cause or another, can evidently result only in increase of the dyspepsia, because, under these circumstances, the process of fermentation above described must be kept up continuously.

Finally, the anatomical alterations undergone by the glandular tissue must be regarded as giving occasion for the occurrence of dyspepsia in the disease under consideration. For the gland tubules becoming in part atrophied by proliferation of the interstitial tissues, and in part fattily degenerated during the progress of the inflammation, the secretory surface becomes perceptibly lessened, and consequently the quantity of gastric juice secreted is decidedly diminished. Under these circumstances the latter no longer suffices for the digestion of the food, and thus indirectly gives occasion to abnormal changes in the ingesta, with consequent dyspepsia.

The same disproportion between the quantity of gastric juice secreted and the amount of food exposed to digestion is found in those conditions known as "*torpidity of digestion*," "*atony of the stomach*," etc. In such cases the gastric mucous membrane reacts too feebly to the stimulus of food, and so does not produce a sufficient quantity of gastric juice. Individuals who suffer from this form of dyspepsia endeavor instinctively to remedy the defect by eating spices and other stimulating articles of diet with their food, and thus to excite a more sufficient production of gastric juice.

Direct evidence of the existence of such pathological conditions may be afforded by the following procedure. If the tube of the stomach pump be introduced into the stomach of a healthy person, the mechanical irritation thus produced, together with the stimulation of cold water poured through the tube, is sufficient to induce a slight secretion from the gastric mucous membrane, and subsequently there flows from the opening of the tube a feebly acid fluid. In torpidity of digestion this slight stimulation is evidently insufficient, for, under these circumstances, *the fluid poured out by the tube is neutral in reaction.*

When, now, from one cause or another, the gastric function becomes permanently perverted, symptoms of dyspepsia appear, among which vomiting is important, because by this means the stomach is emptied of its abnormal contents, the examination of which is most likely to aid in discovering the true nature of the affection.

Aside from those abnormal products of fermentation, the origin of which has been previously discussed, and whose presence in the matters vomited may sometimes be proved chemically by very circumstantial methods, various forms of *fungi* are found on microscopic examination of the *ejecta.* Among these the commonest are the *yeast fungus* (torula cerevisiæ), in alcoholic fermentation, and the *sarcina ventriculi,* known by its quadratic segmentation, which was first discovered in 1842 by J. Goodsir, in the ejecta of a man who suffered from undoubted dilatation of the stomach. These fungi probably bear no relation to the usual fermentative processes.

At least Kühne[1] failed to observe any development of gas, although he allowed sarcinæ mixed with vomited matters to remain for days together over mercury, and also mingled fermentable mixtures of sugar, peptones, saliva, etc., with the fluid containing sarcinæ. R. Weise convinced himself by chemical examination of the sarcina fungus that it contained silicious particles which appeared to be the remains of diatoms; he found always in the same stomach, besides the sarcinæ, certain fungus cells, which he proposes to call *frustulia ventriculi.*

Finally, the masses rejected in that peculiar variety of vomiting called *vomitus matutinus* present an especial form. Since Frerichs (l. c., p. 790) has examined more closely the fluid vomited, which is watery and shows a various reaction—usually alkaline —no doubt can exist any longer that "waterbrash" consists, for the most part, of saliva, swallowed by the patient on an empty stomach, particularly at night, and belched up again in the morning, being found unassimilable. Pyrosis most usually occurs in the chronic catarrh of habitual drinkers, and appears to result from secretion of saliva, dependent (by reflex action) upon the condition of the stomach. Such secretion can at least be induced by irritating the mucous membrane of the stomach, and salivation is encountered clinically, alternating with the vomiting of watery fluid.

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[1] **Lehrb. der Phys. Chemie. S. 59.**

This detailed description of the dyspepsia of chronic gastric catarrh, with its relations to the functions of the stomach and its results, has been deemed advisable, because it forms the most prominent symptom of the affection under consideration, while the various other symptoms are not invariably observed and are of minor significance. For the sake of completeness, however, a short description of the latter will now be given.

When the chyle already undergoing putrefactive change passes on into the intestinal canal, *flatulence* may ensue to a marked degree, and the resulting meteorism may give rise to increased discomfort and constriction of the abdomen. *Constipation* usually exists, occasionally alternating with diarrhœa. *Icterus*, which is sometimes observed in connection with chronic catarrh of the stomach, probably owes its origin to an extension of the inflammation from the mucous membrane of the stomach to that of the duodenum and ductus choledochus.

The urine shows almost constant variations from the normal during the course of chronic gastric catarrh, in the form of deposits rich in phosphates.

The changes observed in the *skin* during the prolonged course of this disease—dryness, roughness, etc., as well as actual eruptions, such as urticaria, eczema, and the like—possess little more significance than the alterations in the urine just mentioned. Quite curious, apparently, is a certain alternation between the affection of the skin and that of the stomach, so that the symptoms of the latter diminish when, for instance, eczema of the face appears (vide W. Fox, l. c., p. 129). These cases, however, are rare, so that Hebra, who frequently observes eczema in connection with dyspepsia, insists that it does not stand in any alternative relation with gastric catarrh; well authenticated cases, like those mentioned, when the two affections appear to alternate, being simply due to coincidence.

Distressing palpitation of the heart and irregularity of the pulse are also observed in cases of chronic gastric catarrh; the *nervous disturbances* connected with this affection are, however, more frequent and of greater interest.

Aside from the headache and vertigo occasionally observed,[1] together with sleeplessness, lassitude, and pains in the limbs, the patient is subject to low spirits, which exercise a depressing influence. Thus, for example, the mind of the rich merchant, as Bird strikingly remarks, may be occupied during his hours of leisure by gloomy forebodings of approaching poverty; the workman sees visions of the almshouse pass before his eyes, while his actual situation is in every respect comfortable and well assured.

We do not at present fully understand the precise relation between digestive disturbances and the nervous system; we cannot, however, ignore the reaction of the latter under the influence of the digestion. The national diet is an index to the tendency of thought in whole nations, and it is a daily experience that during the progress of digestion thought becomes less active and discomfort takes the place of ease. In like manner, the fact observed in lunatic asylums cannot be overlooked, that symptoms of gastric catarrh not unfrequently become developed to a marked degree during the course of those forms of insanity connected with depression of spirits; unusual mental excitement may also cause disturbance of the digestive process. Diseases of the sexual organs, hysteria, and the like, are usually accompanied by dyspepsia. These correlations of the nervous and digestive systems cannot at present be explained; even conjectures upon the subject are out of place, so long as we know little or nothing of the course taken by the secretory nerves of the stomach, and while self-poisoning by abnormal digestive processes is so extraordinarily infrequent. Senator's hypothesis, to which allusion has been made, which suggests that the products of abnormal digestion, butyric acid, and similar heterogeneous substances, may become reabsorbed and act as poisons upon the central nervous system, explains this connection in part, and is therefore worthy of consideration.

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[1] *The vertigo observed as a result of dyspepsia* is undoubtedly in many cases nothing more than a symptom of the anæmia caused by the digestive disturbance; in certain cases, however, it appears to be directly connected with the gastric nerves, and to be designated with propriety, "stomachic vertigo." [The "*vertigo a stomacho læso*" of Trousseau.—TRANS.]. Thus, I was recently consulted by a gentleman forty-three years of age, who had suffered for nine years with symptoms of dyspepsia together with vertigo. The latter never occurred in the morning, but only after meals, particularly in the evening, and while the patient was seated. He was obliged to stand up in order to be rid of the uncomfortable sensation. When he turned a corner, or if he attempted to keep step with any one, he was also attacked by vertigo. This disappeared so soon as the passage of flatus or eructation took place. *The case was peculiarly remarkable from the fact that I could excite vertigo artificially by pressing the patient over the epigastric region while in a recumbent position*, and then allowing him to rise. When he simply lay down and rose again without palpation of the epigastrium having been performed, vertigo did not appear. This accident calls to mind Schützenberger's artificial production of hysterical attacks by momentary pressure upon the ovaries.

The *vigor* and *nutrition* of the patient naturally suffer under the influence of persistent dyspepsia, when the transformation of the ingesta is continually disturbed and resorption hindered, and particularly when a large part of the food taken is rejected again by vomiting. If the expression of hypochondriasis is added to this, the individual presents a prematurely aged appearance. The general nutrition of many patients suffering from chronic gastric catarrh is, however, not materially altered.

The course of the disease is characteristically tedious. If the patient neglect himself, or if the affection is dependent upon incurable disease of other organs, the symptoms of chronic gastric catarrh may persist for years. Even when convalescence has set in, relapses are very common, and exacerbations in the course of the disease are not infrequent. Chronic catarrh of the stomach is not in itself fatal; it may, however, by impairing nutrition, bring about enfeeblement of the organism, and so indirectly shorten life.

Diagnosis.

As persistent dyspepsia is the chief symptom of chronic gastric catarrh, and as it presents itself at the same time with more or less prominence in most other diseases of the stomach, it is evident that the diagnosis usually offers some difficulty. It must, indeed, be admitted, as has already been remarked, that, as a rule, the diagnosis of chronic gastric catarrh can only be established with certainty when the other more severe chronic affections of the stomach, particularly gastric ulcer, carcinoma, and dilatation, can be positively excluded. The essential points that distinguish these conditions from chronic gastric catarrh will be given when we come to consider the diagnosis of the affections in question. In other cases the difficulty lies less in establishing a differential diagnosis between these affections and chronic gastritis, than in ascertaining the original affection from which this has arisen. It should be remarked, in this connection, that, in addition to physical exploration of the thoracic and abdominal organs, examination of the urine should not be neglected;—chronic Bright's disease, particularly contracted kidney, occurring at times with the symptoms of dyspepsia, and often giving occasion to egregious mistakes in diagnosis.

The *diagnosis of dyspepsia* is easy, but its reference to some particular morbid condition in individual instances is usually difficult. The *positive* assertion that chronic gastritis, and nothing else, is present, is often only possible after long-continued observation of the case. Occasionally the appearance of the matters vomited will aid the diagnosis, a considerable admixture of blood pointing strongly towards ulcer or carcinoma; while the presence of quantities of *mucus*, in regard to the appearance and reaction of which some remark has been made above, is an especial attribute of chronic gastric catarrh.

Prognosis.

Chronic gastric catarrh not being directly fatal, the prognosis, so far as life is concerned, is favorable, the disease usually terminating in convalescence. It is, however, beyond doubt a stubborn affection, the cause usually being irremovable, whether an incurable affection, as pulmonary, cardiac, or hepatic disease, or the habit of continually irritating the stomach with ingesta, particularly alcoholic liquors. In either case therapeutic efforts are in vain.

As we find, in all debilitating diseases, *old people* bear the disturbances of nutrition, necessarily connected with chronic gastric catarrh, much less easily than the young, and in the aged the occurrence of this affection undoubtedly hastens death by marasmus. The prognosis is favorable in those cases where the gastric catarrh originates in removable causes, particularly where it has been brought on by improper food, chlorosis, etc.

Treatment.

Close examination of the circumstances connected with the etiology of chronic gastric catarrh fully shows how seldom and with what difficulty the causes of this disease can be removed, or even modified in their action, by medical art. That, however, the treatment of the original disease should be attempted, and that at the same time all known therapeutic measures available against the chronic gastritis should be duly considered, needs hardly to be said.

In that form of gastric catarrh which is dependent upon *pulmonary and cardiac affections*, the fact that *digitalis* causes loss of appetite, nausea, and vomiting, should not prevent the employment of this medicine, for the improvement in the action of the heart brought about by the remedy will be accompanied by a corresponding amendment in the chronic gastritis, since the latter is due to disturbance of the circulation. In those cases where, although digitalis exercises a sedative and strengthening influence upon the action of the heart, the gastritis continues unchecked, I am inclined to attribute the origin of the latter to some other cause than venous congestion.

Gastric catarrh dependent upon congestion of the portal circulation demands consideration of the hepatic affection involved, and may also indicate depletion of the rectal veins, the use of salines, etc.

In the treatment of chronic catarrh of the stomach it is important to know whether an anæmic condition is not at the root of the difficulty. In such cases *hydrochloric acid* may be employed in connection with preparations of iron. It may be administered in doses of from six to eight drops in a wine-glass of water, to be taken a few hours after meals. The rationale of such medication is evident, aside from clinical experience, from the results of Manassëin's experiments (vide supra).

If we have to deal with a gastric catarrh which is the result of chilling of the surface, Turkish baths, cold frictions, and similar hydrotherapeutic methods may be employed. These last, and also the use of an abdominal bandage of flannel, are the best means of guarding against relapse.

In cases where the catarrh of the stomach is dependent upon incurable lesions, as in tuberculosis, carcinoma, old age, etc., or where chronic Bright's disease is the exciting cause of gastric inflammation, or of uræmic dyspepsia resembling it, we can scarcely expect to fulfill the indication of "removal of the cause," nor can we indeed hope for much benefit from therapeutic measures. We can scarcely anticipate more from denying spirits to the habitual drinker, or advising careful attention to diet in the case of poor persons who are restricted to a fare of potatoes and the like.

While then we can only, in rare instances, remove the remote cause of chronic gastric catarrh, yet, fortunately, on the other hand, the proximate *irritation* which keeps up the disease may often be combated successfully by medical means. We have seen that fermentative processes occur within the stomach as a result of the disease, and that the products of this fermentation are of such a nature as to prevent perfect digestion of the food. Now experiments upon fermentation, carried on externally to the organism, show that when this process is once established in the stomach, fresh food introduced into this organ becomes involved in the abnormal transformation. *The first and most important procedure, therefore, in the treatment of well-marked cases of gastric catarrh, is to put a stop to the concomitant fermentation in the stomach.* Various means may be employed to accomplish this end. Either we may make use of medicines calculated to arrest the process of fermentation without removal of the gastric contents, or we may effect the removal upwards or downwards of the decomposing matter which keeps up the catarrh, and thus furnish a clear field for the digestion of food which may subsequently be taken into the stomach.

As regards the first method of treatment, unfortunately we possess no medicine of undoubted *anti-fermentative* action in disturbances of digestion. *Creosote* is the most likely to be useful, or perhaps *benzine*, which Naunyn recommends as more certain than the other. The former may be administered in pill form in the dose of from one-half to three-quarters of a grain, while benzine may be given in twenty-drop doses.

More certain and more correct in principle is the use of the second mode of treatment, *by which the cavity of the stomach is freed from its decomposing contents by removal upwards or downwards.*

Evacuation, by means of the intestine, is best attained by the use of some of the alkaline-saline mineral waters, in which sulphate of soda is the active ingredient. Among these Carlsbad, Marienbad, and Tarasp are chiefly to be preferred; the latter is most useful in cases where excessive flatulence coexists, on account of the notable proportion of carbonates, particularly carbonate of lime, which it contains. *The specific action of the waters of Tarasp* in such cases may probably be explained by the fact that where fermentation, with the development of much gas and acid, occurs, the richness of these waters in the salts mentioned enables them to neutralize the acid, and then *to remove the contents of the intestinal tract, thus somewhat improved in character, before further fermentation can take place to an appreciable extent.* The employment of the natural sulphate of soda mineral water is to be preferred to the prescription of simple Glauber's salts, since we have in the case of these waters, besides the action of this salt, the effect of certain other (sodium) salts upon the gastric mucous membrane. The therapeutic effect of the latter has been demonstrated by long experience, and will be referred to again further on. If, on the other hand, we desire in a given case merely to carry out the indication above mentioned—that is, the removal of the injurious contents of the stomach—we may employ, in place of the natural mineral water, artificial Carlsbad salts (tolerably pure sulphate of soda) dissolved in water. In any case, we must observe this rule in its administration, namely, *to give enough mineral water or salt to produce several watery stools.* I do not consider the administration of other purgatives advisable, since we may attain the desired object by the means above mentioned, without any evil effect. At most it may be recommended, in cases of obstinate constipation with atony of the gastric mucous membrane, to begin treatment with some water rich in chloride of sodium, as, for instance, Mergentheim or Kissingen, instead of Carlsbad.

To empty the stomach *through the mouth* seems, however, much more rational than the method just mentioned. For while purgation involves irritation of the intestinal tract throughout its entire length by the passage of abnormally changed ingesta, evacuation by the mouth not only relieves the stomach of the offending matters, but at the same time removes them from the body. From this point of view, furthermore, that method will deserve most confidence which serves most perfectly to evacuate the stomach, and this object is best attained by means of the *stomach pump.* The thorough unloading and cleansing of this organ, which is effected by the instrument in question, is, as Schliep's recently published researches show, most admirable in its influence upon simple gastric catarrh, and the treatment of such cases should always begin with it. If there are insuperable difficulties in the case of any patient, if, as occasionally happens, fainting supervenes whenever an attempt is made to introduce the tube, or if, in spite of all persuasion, the patient declines to allow the procedure, administration of an *emetic* is indicated. We may employ ipecacuanha, or, better, for the reasons above mentioned (vide Acute Gastric Catarrh), apomorphia. By means of emetics, and still more by the use of the pump, not only may the abnormal fermenting ingesta be removed, but also viscid mucus which adheres to the walls of the stomach and interferes with the action of the gastric juice upon any food which may subsequently be taken.

It would be unreasonable to expect that a *single* evacuation of the stomach by this method would be sufficient, excepting in rare instances, to restore the diseased mucous membrane to a healthy condition, even if it should be protected against further injury. The probability of a cure will be much greater if evacuation by means of the stomach pump be repeatedly practised, or if the Carlsbad water be used during a long period.

After the diseased gastric mucous membrane has been, as it were, prepared for recovery by one of the methods just mentioned, the most important indication is *to preserve it from all irritation* which might interfere with the cure. This indication is only perfectly fulfilled when, for a considerable period, absolutely no food at all is received into the stomach, for even the blandest nourishment excites the activity of the affected mucous membrane, and in this way may disturb the healing process. In severe cases, therefore, it is always advisable to prevent entirely the reception of food by the mouth and to nourish the patient for some time entirely by the rectum.

Among nutritious enemata pancreatic-meat emulsion (vide Gastric Ulcer) is to be preferred. In case pancreatic glands cannot be obtained, enemata of a solution of albumen may be employed. This is prepared "by beating up the white of an egg with one and a half times its volume of water. After the mixture has stood for several hours with frequent stirring, it is to be filtered." Czerny and Latschenberger's researches upon absorption[1] speak in favor of enemata of this description; I myself have had, as yet, too little experience to enable me to form an opinion.

In milder cases, however, it is sufficient to order *an easily-digestible diet.* The question as to the digestibility of any given article of diet may be decided from different standpoints. Recent experiments upon the rapidity with which a portion of the food passes through the stomach and upper portion of the intestinal tract, as well as our knowledge of the absorptive power of the great intestine, impels us to ascribe in general to the latter no small share in the work of digestion. We must not, therefore, allow a one-sided consideration of the action of the gastric juice upon the food to influence us in deciding whether one form of nourishment is more digestible than another. The question must be decided, rather, by estimating the degree to which the food is altered by the digestive juices during its passage through the entire alimentary canal, and, at the same time, the amount of nitrogen, fat, etc., which it yields for the nourishment of the body. When, on the other hand, we come to consider the case of patients *suffering from diseases of the stomach*, our ideas of relative digestibility must be formed from an entirely different point of view. In these cases the most digestible food is the food which passes most quickly from the diseased stomach, which is the softest, which presents the fewest inequalities of surface, etc. In a word, *the less a given food arouses the activity of the diseased organ, the more digestible it is.*

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[1] Virchow's Archiv. Bd. 59, p. 1 et seq. *Wiel*, Tisch für Magenkranke, 1875, s. 64, where allusion is made to Kussmaul's and Wiel's experiments with this solution; nothing is as yet known of the results of these researches.

Desirable as it may be that further researches should be made upon the digestibility of foods in general, from a physiological point of view, we can scarcely hope that much is to be gained by this means towards deciding what nourishment is appropriate for patients suffering from gastric affections. For, apart from the different standpoints, which, as above remarked, we must usually take in deciding upon a dietary in gastric affections, the question of digestibility must, in a given case, depend also upon the idiosyncrasies of the patient, his constitution, habits, opinions, tastes, and the like. Most important, however, is the origin and nature of the gastric trouble, which must, to a great extent, decide the question in any given case. We must, therefore, for the present, fall back almost exclusively upon clinical experience in the choice of a diet in diseases of the stomach; and we are, I think, all the more justified in taking the empirical standpoint, since conclusive scientific researches in this department of physiology are not at present available to any extent; and, on the other hand, whole nations as well as individuals have striven unconsciously for thousands of years and still continue to strive towards the attainment of a perfect dietary.

We shall content ourselves, therefore, with stating briefly those foods which experience has shown to be usually easily digested, and which may be employed in affections of the stomach, and particularly in chronic gastric catarrh.

Foods composed largely of *carbohydrates* are only to be permitted sparingly in diseases of the stomach. It is true that these are easily digestible under ordinary circumstances, since they may be dissolved and absorbed by simply passing through the process of lactic acid fermentation without the aid of the digestive juices, thus demanding a minimum of activity on the part of the digestive organs, but their ingestion in large quantities is however inappropriate in gastric disorders, because they go to form those abnormal products of fermentation mentioned above. They must, therefore, when possible, be omitted entirely from the bill of fare in diseases of the stomach, or may at most be permitted only in mild cases. They are as follows: a little white bread, biscuit, rusk; among vegetables, the most tender, such as asparagus, young hops, hulled sweet peas, young carrots, potato soup; in any case these should be eaten of but sparingly.

Patients suffering with chronic gastric catarrh should not be permitted to eat *fats*. In addition to the fact that the gastric juice only penetrates with difficulty morsels of food which are enclosed in fat, and that in consequence these do not undergo "preliminary digestion" in the stomach, fatty acids may be formed from the fat within the stomach and contribute to the sour, rancid eructation, heart-burn, etc.

From these facts, therefore, we must conclude that in diseases of the stomach, neither carbohydrates nor fats form appropriate nourishment, and that the only question is whether the *albuminoids*, in the shape, for instance, of substances rich in albumen, may be employed, without further change, as food in these affections. *À priori* the albuminoids should be inferior to the carbohydrates in digestibility, for while the latter do not ordinarily require the aid of the digestive juices for their transformation and absorption, the digestion of albuminoids without this is impossible. On the other hand, however, the albuminoids do not so easily undergo abnormal decomposition, and their digestibility may be decidedly increased by appropriate methods of preparation. Furthermore, some albuminous substances are much more digestible than others.

As regards the use of *eggs*, I permit the patient to eat them only when soft-boiled, since hard-boiled or raw eggs must be looked upon as difficult of digestion.

Meat should, in general, not be prepared when too fresh, since the gradual increase of acidity which it undergoes upon keeping aids the action of the pepsin, and, in particular, begins the conversion of the gelatigenous materials into gelatine, which process can then be more easily carried out in the stomach. Among the most digestible foods are the white meat of fowls, the glandular organs of young animals, the flesh of young pigeons, the thymus gland of the calf, etc. The digestibility of the latter is probably due to the fact that its connective tissue is partly changed into gelatine in the process of cooking (Brücke). The flesh of fishes, also, when boiled and free from fat, appears to be easy of digestion, in spite of the general opinion to the contrary—so, at least, Beaumont's table would indicate, for his experiments upon St. Martin showed fish to be the most digestible of all meats. I also usually permit the use of boiled veal in affections of the stomach. Many patients cannot deny themselves the use of beef; I permit this only when roasted rare, remembering the old rule that too much roasting, like too much boiling, makes meat tough—a rule which receives scientific support from the experiments of Fick, who found that boiled meat takes three times as long as raw meat to dissolve in the same digestive fluid. Meat, like all other food, should be chewed into small pieces before swallowing, and, for the reasons given above, no greasy sauce should be eaten at the same time.

Milk, as a peculiarly digestible form of nourishment, and as one usually regarded as the most natural, demands especial mention. Its easy digestibility, a generally recognized quality, depends upon the fact that the caseine, which is precipitated in the stomach, is easily soluble, and its soft floccules cannot wound the mucous membrane. In addition, certain portions of the milk pass quickly through the stomach. As is well known, milk is excellently well borne in most cases of gastric disease; yet there are cases in which the caseine coagulates with unusual solidity, and is difficult of solution. In the case of many patients, moreover, milk ferments very rapidly in the stomach, and to an extreme degree, probably for the same reasons and under the same abnormal circumstances as those which cause the lactic acid fermentation of starch in the stomach to be more rapid and extensive than usual (vide supra).

Since, then, even so easily digestible a food as milk is badly borne at times, we are occasionally forced to make use of artificial nourishment in the form of a preparation which may be considered even more easy of digestion than the normal food, because it has previously been subjected artificially to partial digestion outside of the body. I refer to the *solution of meat* devised by J. Rosenthal and myself, regarding the value of which as a means of nourishment for patients with gastric trouble a more detailed account will be given under the head of Gastric Ulcer, for the treatment of which disease it is particularly well suited.

In a small number of cases, particularly in *atony* of the gastric mucous membrane, easily digestible nourishment is of no use, but, on the contrary, is rather harmful. In order to prevent, so far as possible, the occurrence of fermentation in such cases, we must confine the patient to a meat diet, as in other forms of chronic gastric catarrh. It is unnecessary, however, to be so particular respecting the especial sort of food as in the other varieties. When, in this form of the disease, the patient who suffers from insufficient secretion of gastric juice has accustomed himself to the habitual use of sharp spices and similar gastric stimulants, these are to be withdrawn very gradually, or replaced by harmless substances, calculated to induce a freer secretion on the part of the mucous membrane. Among the latter may be mentioned *cold* water (ice), *alkalies*, usually prescribed in the form of Ems or Luhatschowitzer mineral water, or the so-called *bitters*.

The action of the bitters is a complicated one. In the first place, they cause the saliva to be more abundantly secreted. This is followed by an increased secretion of gastric juice, the saliva which is swallowed arousing the gastric mucous membrane to energetic action. Then, too, as H. Köhler's[1] researches go to show, the increased blood pressure induced by these substances in all probability also increases the secretion from the gastric mucous membrane. Finally, the bitters may perhaps also be regarded[2] as anti-fermentative in their action. Among the bitters, *quassia*, *calamus*, *gentian*, *cetraria*, *columbo*, etc., are particularly to be recommended. *Strychnia*, in other respects so totally different from the generally harmless bitters, must also be regarded as a stomachic.

Red wine of some kind, mingled with *water*, is perhaps the best *beverage*, though it is better for the patient to abstain entirely from drinking alcoholics. Where wine is imperatively demanded, it may be administered per rectum. It is highly reprehensible to accede to the patient's pressing request and to allow the use of light beer or large quantities of some acid wine. Should intense thirst be experienced, small morsels of ice may be swallowed; these tend to allay thirst and at the same time excite the secretion of gastric juice.

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[1] Handb. der physiol. Therapeutik, etc. 1875. p. 142.

[2] Ibidem, p. 144.

In connection with the regimen just recommended, the indication demands the use of *alkaline mineral waters.*

The *carbonate of soda* contained in these waters seems to exert a double influence, neutralizing the excess of acid in the stomach, while at the same time—which is of more importance—it excites the diseased mucous membrane to the secretion of gastric juice. Such at least would appear to be the case from our experiments upon the introduction of solutions of soda into the stomachs of dogs in whom gastric fistula had been established. The effect of the essentially alkaline waters—Vichy, Geilnau, etc.—is in accordance with this theory.

The action of those alkaline waters which contain *chloride of sodium* as well as carbonate of soda is apparently still more energetic, and their use is particularly to be recommended in the atonic forms of gastric catarrh. The warmer among these springs, Ems and Royat, act more mildly, on account of their higher temperature, than the colder ones, such as Luhatschowitz, Tönnisteiner, Heilbrunnen, etc.

To the action of the two compounds just mentioned, must be added that of the *sulphate of soda* contained in the waters of Carlsbad, Tarasp, and other localities, so celebrated for their beneficial action in chronic gastric catarrh. The chief effect of these waters is to increase peristaltic action, which they arouse to a more marked degree than do those previously mentioned. Allusion has already been made to the necessity of this.

Finally, in estimating the general effect of these various waters, the quantity of *free carbonic acid* which they contain must be taken into consideration. It is generally conceded that the contact of this gas with the mucous membrane of the stomach excites the secretion of gastric juice, that it brings up with it, in the eructations which it causes, the various gases evolved in the stomach by the fermentative process, and that it induces a feeling of warmth and relief.

It seems to me, however, after all, that it is impossible, as yet, to establish more precise indications for the use of the different alkaline mineral waters. The general principles which must be kept in view when ordering a course of these waters have been enunciated with sufficient clearness, I think, from time to time in the foregoing pages. The prominence of one or another symptom in the course of the disease, the constitution and external circumstances of the patient, etc., will decide in any given case the particular mineral water to be employed.

In accordance with the principles alluded to, *hydrochloric acid* is indicated in most of those cases where, in addition to the regimen above described, still further treatment seems desirable; while the administration of *pepsin* would be useful only when, in spite of the introduction of muriatic acid, the process of digestion goes on too slowly and unsatisfactorily, or when the secretion of gastric juice is decreased *in toto*, as in atony of the gastric mucous membrane. Possibly such a condition underlies the gastric catarrh of scrofulous children, in which W. Fox particularly recommends the employment of pepsin. Since the "French" pepsin is a mixture of pepsin, peptones, and lactic acid, its action in many cases of gastric catarrh may in all probability be regarded as that of an acid as well as a ferment.

When individual symptoms become particularly prominent in the course of the disease, or when the affection fails to yield to the remedies above enumerated, an especial course of treatment directed against these symptoms is demanded.

Excessively acid eructation and the *heartburn* connected therewith call for the use of antacids; but these must not be given in too large doses or for a prolonged period. The advantage gained by their use is only transitory, and, as was remarked when treating of fermentation, neutralization of the gastric juice by these substances often does more harm than good.

Slight *pain* and dull sensation of pressure seldom call for therapeutic interference. Laurel water with morphia, a few leeches, stimulation of the skin to a greater or less degree by the ordinary methods, these form the usual means to be employed under such circumstances. Bismuth and nitrate of silver, which were originally employed in cardialgia, are also to be recommended in chronic gastric catarrh, as well as in other diseases of the stomach. The former remedy (in the shape of *subnitrate of bismuth*) is, according to Fox, "the most efficient, as a rule, of the different remedies." Yet Fox himself administers this substance, sometimes in connection with magnesia, sometimes with morphia or hydrocyanic acid, and finds the indication for its use partly in the different aspect of the tongue. Besides this, in spite of the very general use of the remedy, it is not yet clearly established in what class of cases bismuth is to be used to advantage. Upon the whole, then, it would seem advisable to have recourse to it only when a regularly planned course of treatment does not succeed and we have to fall back upon experiments.

Vomiting must disappear with the other symptoms of gastric catarrh under the use of the remedies just enumerated. The rapid disappearance of this symptom as a result of the use of the stomach pump is surprising. Should, however, this symptom have to be especially combated, the usual means, bits of ice, opium, etc., will prove sufficient.

In order to remove *constipation*, the use of Carlsbad, Kissingen, Ragoczy, or some of the natural mineral waters of this kind above mentioned, will usually be sufficient. The effect produced by the stomach pump may also render the use of a purgative unnecessary. In cases of obstinate constipation, or where this returns after a course of Carlsbad water, purgatives may be employed. Rhubarb pills are usually chosen (as the compound rhubarb pill); or, in order to act upon the large intestine alone, aloes, or extract of colocynth, may be employed.

Finally, for the purpose of hastening *convalescence*, it is advisable, besides an easily digestible, strengthening regimen, to allow the patient who has been suffering from chronic gastric catarrh to use some ferruginous mineral water, as that of Pyrmont, Schwalbach, or Spa. Sea-bathing is also to be particularly recommended in that torpor of the gastric mucous membrane which follows convalescence.

Ulcus Ventriculi Simplex (Cruveilhier).

Syn.: Ulcus rotundum, perforans, chronicum, corrosivum, gastric ulcer.

Ulcer of the Stomach (in the strict sense).

Syn.: Round, chronic, etc., ulcer of the stomach.

Galen, De loc. affect. Lib. V. Cap. 6.—*Celsus*, De medic. Lib. IV. Cap. 5.—*Grassius*, 1695, Ephemerid. German. a. III. 1696. observ. 40.—*Littré*, Acad. Royale des Sciences de l'année 1704. Hist. pag. 36.—*van Swieten*, 1754, Comm. in Boerhaave Aphorism. Tom. III. § 955. S. 149.—*Odier*, Von d. antispasmodischen Wirkung des Magisterium Bismuthi. Sammlung auserl. Abh. XII. 326. 1786.—*M. Baillie*, Morbid anatomy, etc. 1793. p. 87.—*X. Müller*, De usu argenti nitric. praes.—*Autenrieth*, Diss. Inaug. 1829. Tübingen.—*J. Abercrombie's* Pathological and practical researches on diseases of the Stomach, etc. 1832.—*Cruveilhier*, Anatomie pathologique. 1829-1835. Livraison X.—*Idem*, Comptes rendues. Janv. et Mars. 1856.—*Rokitansky*, Med. Jahrb. des k. k. österr. Staates. 1839. S. 184.—*Idem*, Lehrb. der pathol. Anat. 3. Aufl. 1861. Band III. S. 165.—*Andral*, Clinique médicale. 1839. II. S. 102.—*Jaksch*, Beitrag zur Lehre vom perforirenden Magengeschwüre. Prager Vierteljahrschr. III. S. 1. 1844.—*Osborne*, Propositions, etc., Dublin Journ. of Medic. Science. 1845. vol. 27. p. 357.—*Günzburg*, Zur Kritik des Magengeschwürs. Archiv für phys. Heilkunde. 1852. S. 516.—*Virchow*, Historisches u. s. w. zur Lehre der Unterleibsaffectionen. His Archiv. V. S. 281. 1853.—*Papellier*, Diss. Inaug. Erlang. praes. *Dittrich*, 1854, Tuberc. u. Magengeschwür.—*Willigk*, Sectionsergebnisse. Prager Vierteljahrschr. 1856. Band 50 und 51.—*Plange*, Diss. Berol. 1859.—*L. Müller*, Das corrosive Geschwür im Magen und Darmkanal, etc. Erlangen. 1860. The best monograph, with an accurate bibliography.—*Traube*, Ein paar Bemerkungen zur Lehre vom einfachen Magengeschwür. Deutsche Klinik. 1861. No. 10. Ges. Beitr. II. 531.—*Brinton*, Diseases of the stomach. 1859.—*Panum*, Experim. Beiträge zur Lehre von der Embolie. Virchow's Archiv. 25. 1862.—*Förster*, Handbuch der spec. pathol. Anat. 1863. S. 87.—*Bamberger*, Krankheiten des chylopoëtischen Systems. 1864.—*G. Merkel*, Casuistischer Beitrag zur Entstehung des runden Magen- and Darmgeschwürs. Wiener med. Presse. 1866. Nos. 30 u. 31.—*Idem*, ibid. 42 u. 43.—*Idem*, ibid. 1869. No. 39.—*Ebstein*, Complic. der Trichinose mit d. corrosiven Magengeschw. Wiener medic. Presse. 1866. No. 12.—*Idem*, Virchow's Archiv. 40. 1867.—*Klebs*, Handb. der pathol. Anat. 1868. S. 181.—*Steiner*, Diss. Berolin. 1868. praes. *Virchow*.—*Wollmann*, Diss. Berol. 1868. praes. *Virchow*.—*Pavy*, On gastric erosion. Guy's Hospital Reports. 1868.—*Fenwick*, The morbid states of the stomach. 1868. Cap. 15.—*Gerhardt*, Zur Aetiologie und Therapie des runden Magengeschwürs. Wiener med. Presse 1868.—*M. Roth*, Experim. über Entstehung des runden Mg. Virchow's Archiv. 45. S. 300. 1869.—*Axel Key*, Om det corrosiva magsårets u. s. w. Hygiea. Referat in

Gurlt-Virchow's Jahresber. 1871.— *W. Müller*, Jenaische Zeitschrift. V. 2. S. 175. 1870.—*F. Starke*, Mittheilung über das chron. Duoden.- u. Mggschw. Deutsche Klinik. 1870. 26–29.—*H. Ziemssen*, Ueber d. Behandlung des Magengeschw. Volkmann's Sammlung klin. Vorträge. No. 15. 1871.—*Rindfleisch*, Lehrb. der path. Anat. 1871. S. 316–319.—*Ross*, Lancet. 1871. Jan.— *Tinley*, ibid. April.— *Wilson Fox*, The diseases of the stomach. 1872. p. 146.—*P. Schliep*, Zur Behandlung mit der Magenpumpe. 1874. Arch. für klin. Medic. Bd. XIII. S. 453.— *W. Ebstein*, Experimentelle Untersuchungen, etc. Archiv. für experimentelle Pathol. u. Pharmakologie. II. 2. 1874. S. 181.—*A. Böttcher*, Zur Genese des perforirenden Magengeschwürs. Dorpater medic. Zeitschrift. 1874. V. Band. Heft 2. S. 148.

Galen mentions ulcer of the stomach, and Celsus lays down rules for its treatment, which are generally accepted, even at the present day: "Adhibendi lenes et glutinosi cibi sed citra satietatem; omnia acria atque acida removenda; vino utendum, sed neque praefrigido neque nimis calido."

As autopsies came to be made more frequently, cases were occasionally observed in which death was caused by perforation of the ulcerated stomach. Thus, Grassius, in 1695, gives an account of a perforated gastric ulcer, which, being obstructed by the spleen, allowed but a small amount of food to escape from the stomach into the peritoneal cavity. Ere long fatal hemorrhages also came to be recognized as due to the ulceration in the stomach. In 1704 Littré found the source of a severe fatal gastro-intestinal hemorrhage in an "ulcus rotundum," five lines broad and half a line deep.

In his work on pathological anatomy, Matthew Baillie devotes a special chapter to the subject of gastric ulcer, of which he gives what for the time was an excellent description. "Opportunities occasionally offer themselves of observing ulcers of the stomach; these sometimes resemble common ulcers in any other part of the body, but *frequently they have a peculiar appearance*." He speaks also of their "regular" edges, and of the healthy appearance of the gastric mucous membrane in the neighborhood of the ulcer; it seems "as if some little time before a part had been cut out from the stomach with a knife." "These ulcers sometimes destroy only a portion of the coats of the stomach at some one part, and at other times destroy them entirely," etc.

An important service was rendered to the *symptomatology* of this disease by the excellent reports of cases by J. Abercrombie, which are still worth consulting at the present day. A strict separation between what we understand as simple ulcer and cancerous ulceration was, however, as yet impossible. This distinction was first made by Cruveilhier, who defined the "ulcus ventriculi simplex," as he called it, as a distinct affection, and to him we are mainly indebted for the views at present entertained in regard to this disease. He called special attention also to the curability of the disease, and laid down the rules for its rational treatment. This work, which marks an epoch in the study of this affection, was shortly afterwards followed by Rokitansky's admirable treatise on the same subject.

What has since been written on gastric ulcer relates merely to the more minute details of the *symptomatology*, *treatment*, and *pathogenesis*. The most zealous inquiry has been devoted for a long time to the causation of the disease, but as yet without conclusive results. Virchow, especially, has broken ground for future inquiry by referring the origin of gastric ulcers to derangements of circulation in the gastric vessels. For the most industrious and most comprehensive monograph on ulcer of the stomach and duodenum we are indebted to L. Müller; finally, among recent English writers, Brinton has produced much that is original upon this as well as upon other diseases of the stomach.

Pathological Anatomy.

Gastric ulcer is a specific variety of ulcer, which is met with not only in the stomach, but also in the duodenum and terminal portion of the œsophagus, and which has only one analogue, viz., the corroding ulcer of the neck of the uterus. The *form* of the ulcer is that of a funnel, the loss of substance being greater upon the mucous membrane than in the external layers, so as often to give the ulcer a striking gradient appearance. In older ulcers, however, the walls are perpendicular. The periphery is usually circular (ulcus "rotundum"), but large ulcers are frequently elliptical, and in very rare cases several of them, as, for instance, at the pylorus, run together, so as to assume the form of a girdle. The edges are sharply cut, looking in exquisite cases "as if a piece of the wall of the stomach had been removed by a punch" (Rokitansky). When the ulcer is of recent origin it is characterized by an absence of all inflammatory infiltration in the neighborhood, but usually the edges are found hard and thickened; this condition has been regarded by Cruveilhier as an indication of the chronicity of the ulcer. At all events, in very acute cases there is no surrounding inflammation; cases do occur, however, in which the granulation tissue is so much developed as to make it difficult to decide whether the disease be a simple ulcer or a carcinoma granulosum (*e. g.*, see the case of W. Müller). Beyond the immediate neighborhood of the ulcer the mucous membrane is usually normal, but in some cases there are signs of acute or chronic gastritis.

The *base* of the ulcer presents different appearances, according to the depth to which the destructive process has advanced;

being formed sometimes by the submucosa, sometimes by the muscular layer, or, finally, by the peritoneal coat. The latter, even after rupture, may still continue to form the floor of the ulcer by the conversion of the peritoneum occupying the lumen of the ulcer into a yellowish crust. Should the ulcerated part of the stomach become adherent to the neighboring organs before perforation of the peritoneum takes place, the base of the ulcer will be formed by the substance of these organs.

The *size* of the ulcer is very variable—rarely smaller than a ten cent piece. The largest ulcer known, that described by Cruveilhier,[1] was six and a half inches long, and three and a third inches wide.

As regards its *location*, the ulcer notoriously occurs more frequently on the posterior than on the anterior wall of the stomach. Brinton, who has compiled a larger number of cases than any other writer (220), states that the posterior surface is affected in about forty per cent. of the cases. Rokitansky's experience shows that the ulcer is almost always situated near the lesser curvature, and frequently directly upon it. This fact, in connection with Brinton's statistics, that fifteen per cent. of the ulcers occupied the pyloric extremity, and twenty-five per cent. the lesser curvature, gives the remarkable result that *in four-fifths of the cases the ulcer is situated upon a region bounded by the posterior wall, the lesser curvature, and the pyloric region.*[2] The rest of the surface of the stomach—the fundus, the anterior surface, the greater curvature, and the cardia—while it is much larger, appears to be affected in only one-fifth of the cases (anterior surface, about five per cent., and the greater curvature and cardia two per cent. each).

Usually, the ulcer is *solitary*, but in about twenty per cent. of the cases two or more ulcers are formed ; occasionally as many as five.

As met with in the cadaver, the ulcer is either open, or else partially or completely cicatrized—the relative frequency of the two conditions being about equal.

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[1] Traité d'Anat. descript. 1852.

[2] Cruveilhier's experience on this point accords entirely with the statements of Brinton.

Thus the results of 11,925 autopsies, compiled partly from Chambers, Gairdner, Dittrich, Jaksch, and others (see Brinton), and partly from the most recent writers, Starke, Steiner, and Wollman, show: open ulcers, 278; cicatrized, 265.

Cicatrization may take place in any stage of the ulcerative process, even after complete perforation of the wall of the stomach, if the perforated spot have been blocked up by adhesions to the adjacent points. The cicatrices of the smaller ulcers generally have a radiate appearance, produced by the surrounding mucous membrane being drawn into folds towards the somewhat depressed cicatrix. The more extensive ulcerations give rise to cord-like cicatrices, which produce more or less narrowing of the stomach (stenosis of the pylorus, hour-glass contraction, etc.).

Gastric ulcer manifests, moreover, a decided tendency to extend in depth (ulcus "perforans"), and ultimately to *perforate* the wall of the stomach, if this event be not prevented by the occurrence of cicatrization. When perforation does take place, it would almost always be followed by a general fatal peritonitis, were it not that fortunately in about forty per cent. of the cases this result is obviated, or at least delayed, by adhesions between the stomach and neighboring parts. While the ulceration is advancing towards the peritoneum, the latter becomes locally irritated and inflamed, and a partial peritonitis results with adhesion of the inflamed spot to the part adjacent. In this way the stomach at the point of ulceration becomes glued to the pancreas, spleen, liver, etc. The active movements of the stomach naturally tend to prevent the occurrence of adhesion, and this is the case particularly with the establishment of a firm connection between the anterior wall of the stomach and the abdominal wall; if union be effected in this case, it consists only of a few fragile threads. Hence the comparatively *greater danger of ulcers situated on the anterior wall of the stomach.*

In the organ, which has thus become adherent to the stomach, and which now forms the base of the ulcer, an inflammatory proliferation of the connective tissue takes place (thus in the liver an interstitial hepatitis), the products of which penetrate deep into the otherwise healthy parenchyma in the form of thick bands of connective tissue. In the most favorable cases cicatrization may even now take place, but in other instances the recession of the obstructing organ produces the formation of cavities, which communicate with the stomach through an aperture at the point of perforation. In still other cases, the ulcerative process extends to the organ overlying the stomach, and gives rise to the formation of external fistulæ, perforations of diaphragm, disintegrations of the liver and spleen, abnormal communications between the stomach and transverse colon or the lower part of the duodenum, etc.

In the worst event, the "cicatrix of adhesion" undergoes rapid necrosis, rupture takes place, and the contents of the stomach escape into the free cavity of the abdomen. This result is, of course, the more likely to happen, the more incomplete the adhesion between the wall of the stomach and the neighboring parts. The result of the perforation is a general peritonitis, which is almost invariably fatal.

Death may be produced also by *hemorrhages* arising from erosions of the larger vessels. Large vessels are, however, involved in the necrosis only when the ulceration extends completely through the wall of the stomach, because the large arteries, such as the splenic, gastric, gastro-epiploic, etc., are too deeply situated to be affected by a superficial erosion. Another occasional source of severe hemorrhage, as Andral particularly has pointed out, is the distended varicose *veins* in the neighborhood of the ulcer, just as we see similar hemorrhages in the neighborhood of chronic inflammatory foci, such as old ulcers of the foot.

Relapses of the disease are not infrequent, even after complete cicatrization of the ulcer has taken place.

Gastric ulcers are very apt to be accompanied by diseases of the circulatory apparatus, endocarditis, endarteritis, etc. (according to Steiner in two-thirds of the cases). The next most common complication is disease of the respiratory organs, especially tuberculosis of the lungs, which occurs, according to the older writers, in about twenty per cent. of the cases, but according to recent reports still more frequently. Pleuro-pneumonia is equally common, while syphilis and carcinoma are rarer complications. The latter disease sometimes seems to locate itself upon the very site of a former ulcer. This point will be considered more fully in the article on Cancer of the Stomach.

Gastric ulcer is a common affection. Out of 13,605 autopsies,[1] open ulcers, or cicatrices resulting from them, were found in 653, giving an *average frequency of not quite five per cent.* The disease seems to be more common in some regions than in others; thus the averages given by the English physicians fall below four per cent., while the reports of the Pathologico-Anatomical Institute at Jena (Starke, W. Müller) give from ten to eleven per cent. It is probable, therefore, that local influences of quite a definite character play a part in the production of gastric ulcer, a point which will be considered more fully in the section on etiology.

Etiology.

The fact that this variety of ulcer, which is characterized by a simple progressive molecular necrosis without suppuration, is encountered only in the stomach or in its immediate neighborhood, has naturally led to an attempt to explain its immediate causation by a corrosive action of the gastric juice. In the chapter on physiology we have seen that under normal circumstances a digestion of the wall of the stomach by the gastric juice is out of the question, except so far as the uppermost acid-reacting layer of the mucous membrane is concerned; because the layer corresponding to the base of the gastric follicles has an alkaline reaction, and digestion by the gastric juice is possible only when the reaction is acid. In order, therefore, to allow the destructive action of the gastric juice to penetrate beyond the superficial layer into the deeper tissues, the normal alkaline reaction of the latter must first cease. This is possible under two conditions; *either the gastric juice becomes abnormally acid*, so that the alkalescence of the wall of the stomach, although continually renewed by the circulation, is no longer able to neutralize the abnormally acid gastric juice, which is thus permitted to penetrate more deeply; *or, on the other hand, the alkalescence of the wall of the stomach is diminished*, so that now even a normal degree of acidity is sufficient to convert the alkaline reaction into an acid one, and to digest the part of the wall affected.

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[1] See the above compilation of post-mortem examinations; also the reports of *Plange* and *Ziemssen.*

The correctness of these assumptions is confirmed by the experiments of Pavy. According to this writer, if a certain quantity of acid be introduced into the stomach, and the circulation be left undisturbed, the stomach remains unaffected; but if with the same amount of acid, the circulation be interrupted, the stomach becomes digested. If the quantity of acid be increased, without interruption of the current of blood, digestion will likewise take place. If we analyze the causes of gastric ulcer in the light of these physiological experiments, and in the light thrown upon the subject by the mass of recorded observations which we possess, the various views expressed by different writers may be summed up under the two following heads:

1. *Diminution of the normal alkalescence of the wall of the stomach as a cause of gastric ulcer.*

Since the alkaline reaction of the deeper layers of the mucous membrane is ultimately conditioned by the distribution of the blood-vessels, every interruption in the circulation must diminish the alkalinity of the obstructed part, and thus make it possible for the acid gastric juice to penetrate more deeply. As long as twenty years ago Virchow called attention to the important part which *derangements of circulation* play in the production of the gastric ulcer, and stated that his observations had led him to the conclusion that "the interruption of the circulation is for the most part due to morbid conditions of the gastric vessels, and particularly to a hemorrhagic necrosis of the mucous membrane." Diseases of the coats of the vessels, obliterations of the arteries, disturbances in the portal circulation, and even strong spasmodic contractions of the stomach must consequently be regarded as causes of hemorrhagic erosions and bleeding ulcers. These views of Virchow have met with general acceptance up to the present time.

The hypothesis, so plausibly stated by this writer, is moreover confirmed by the results of post-mortem examinations, and by experiments. Thus, Steiner found the blood-vessels diseased in two-thirds of the cases in which the autopsy revealed the presence of a gastric ulcer ; in a case of Merkel's, atheroma of the aorta and emboli from this source were discovered in several of the arteries of the body, among others in a small artery of the duodenum, traversing the base of a round ulcer in that locality. So, also, in addition to the ordinary hemorrhagic infarctions, Rindfleisch and others have met with instances in which the infarctions were so much eroded by the gastric juice as to present the appearance of a gastric ulcer. Atheromatous, fatty, and amyloid degenerations have also been found.

But besides the autopsical evidence, that gastric ulcer may be induced by stases in the gastric circulation, direct proof has been afforded by the experiments of Panum, who succeeded in producing hemorrhagic infarctions and ulcers of the gastric mucous membrane of dogs, by introducing substances into the arteries ; while L. Müller, by ligating the vena portæ in rabbits, obtained extravasations of blood and ulcers in the stomach. Very recently Ebstein succeeded, as did Schiff also in part at an earlier date, in artificially exciting ecchymoses and ulcerations in rabbits and dogs, by wounding certain parts of the brain and spinal cord, by irritating sensitive nerves, and by strychnine poisoning, all of these measures being supposed to act chiefly by producing an *increased blood pressure.*

While there can be no doubt, therefore, at the present day, that vascular disturbances in the wall of the stomach may give rise to gastric ulcers, there is still a difference of opinion among writers in regard to the peculiar mode in which these disturbances operate. Although the majority, following the example of Virchow, regard the occlusion of an arterial branch, and therefore *thrombotic* and *embolic* processes, as the most frequent cause of the ulceration, and make the extent and funnel shape of the primary loss of substance dependent upon the size of the area of distribution of the obstructed artery ; others, on the contrary, maintain that this mode of origin is at least extremely rare, because, as a matter of fact, such vascular lesions have been discovered in only a few instances, while in many cases of multiple embolism the gastric arteries have been the very ones to escape occlusion. Moreover it seems to me that the great abundance of blood-vessels in the wall of the stomach, and the favorable arrangement of the anastomoses, make it very improbable that, in the digestive tract, embolism should be followed by the formation of an infarction or by necrobiosis, except when the embolism is very extensive, as may have been the case in Panum's experiments.

The fact that, besides the smaller "hemorrhagic infiltrations" and "erosions," actual ulcers of the stomach have been found in the process of formation, has induced some writers, as Rindfleisch, to regard the origin of the perforating ulcer as identical with that of the so-called hemorrhagic erosion—a view which Rokitansky is known to have expressed previously, and which, as we shall see when we come to explain more fully the formation of the hemorrhagic erosions, is certainly very plausible.

Klebs, while holding to the opinion that gastric ulcer is connected with a pathological condition of an arterial vascular territory, rejects embolism as the most frequent cause, in view of the lack of satisfactory anatomical evidence, and adopts the opinion that the ulcer originates in the spasmodic contraction of one of the areas of arterial distribution. According to this view, therefore, the spasmodic (cardialgic) contraction of the wall of the stomach is the primary disturbance, and the erosion of the now anæmic district by the gastric juice is the secondary lesion. Moreover, after the destruction of the surface has once commenced, the irritant action of the gastric juice upon the denuded ends of the arteries would excite contraction of the same, and the anæmia thus induced would favor the further penetration of the erosion. Similar to this explanation is the one very recently advanced by Axel Key for the genesis of gastric ulcer. He also regards the violent contractions of the muscular coat as the primary condition, but, in his opinion, it is the veins and not the arteries, which are compressed, and thus give rise to the distention of the capillaries, the bleedings, erosions, and ulcerations. Both of these explanations, it seems to me, are equally open to the objection that in chlorosis, hysteria, etc., they presuppose the occurrence of spasm in a stomach which as yet has undergone no anatomical change—a supposition which is purely hypothetical. It is difficult, moreover, to see how a spasm can be so severe and long continued as to permit a digestive action to take place, or how upon this hypothesis we can explain the localization and isolation of the ulcer. Still, it is not to be denied that, in the presence of *other conditions, favorable* to the occurrence of hemorrhagic infiltrations, erosions and ulcerations, spasmodic contractions of the wall of the stomach may accelerate these processes.[1]

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[1] See the article on the Etiology of Hemorrhagic Erosions.

I think we shall be nearer the truth if, instead of insisting upon a particular mode of vascular disturbance as the only plausible explanation, we adhere to the original general proposition of Virchow, viz., that *gastric ulcer is due to a great variety of causes, all of which act by interrupting the circulation in circumscribed portions of the wall of the stomach.* Among the conditions, therefore, which act as either direct anatomical or as predisposing causes in the genesis of the ulcer, we may enumerate thromboses in atheromatous, fatty, or otherwise degenerated gastric arteries, extensive emboli in the same—both of which causes are certainly rare—hemorrhagic infiltrations of the wall of the stomach, and in connection with these chronic hyperæmia of the mucous membrane, violent acts of vomiting, spasmodic contractions of the gastric wall, etc. Upon the whole, however, it must be acknowledged that no one of these causes is predominant in the genesis of gastric ulcer, and that our entire knowledge of the subject is at present limited to isolated facts. Still the views expressed above harmonize so well with what we know of the etiology of the affection, that they rise above the level of mere conjectures. We cannot, however, dismiss the question of pathogenesis without referring to the second condition under which circumscribed self-digestion of the surface of the stomach may take place.

2. *Abnormal increase of acidity of the gastric juice, with normal alkalescence of the gastric mucous membrane, as a cause of simple ulcer of the stomach.*

It is self-evident that when the entire surface of the stomach is in a *normal* condition, an excessive acidity of the gastric juice would have a corrosive effect upon the whole wall of the stomach, or at least upon the more or less considerable portion of the same, with which the acid contents of the stomach came into prolonged contact. If an increased acidity of the gastric juice is to be made responsible for the production of the gastric ulcer, it must be by supposing that the abnormal increase of acidity of the gastric juice is limited to a small district. This might happen when the empty stomach receives ingesta of a character likely to excite severe local irritation of the mucous membrane, and when the additional acid produced by the continuance of the irritant action is not removed from the spot. There is no experimental evidence that such a state of things is possible, and yet the fact that ulcers are specially apt to occur in dram-drinkers, and in cooks who swallow hot food, is suggestive of such a mode of origin.

When an abnormally acid gastric juice attacks an *already injured* portion of the gastric mucous membrane, the denuded ends of the arteries, as Klebs has described in detail, may be excited to contraction, and in this way a local anæmia may be produced, which, as was previously mentioned, is favorable to the occurrence of erosion. *Such a concurrence of the two conditions predisposing to erosion—anœmia and a temporarily increased acidity of the gastric juice—must be inferred in the case of chronic gastric ulcer*, because otherwise it would be impossible to explain why, for example, traumatic solutions of continuity in the wall of the stomach heal rapidly, although vascular disturbances evidently occur in the surface of the wound, while the course pursued by the chronic gastric ulcer is entirely different.

This is the explanation also of the fact mentioned elsewhere, that simple catarrhal ulcers and diphtheritic patches on the wall of the stomach may be converted, under certain circumstances, into corroding ulcers.

The most recent hypothesis, in regard to the origin of gastric ulcer, is that proposed by A. Boettcher. In some autopsies made by him he found the borders and bases of the ulcers penetrated with masses of micrococci, and for this reason he infers that at least a part of the perforating ulcers of the stomach and duodenum are of parasitic origin.

The indirect causes of gastric ulcer are not much better understood than is its direct origin.

In general it appears that the *female* sex is more apt to be affected than the male. Willigk found the ratio to be three to two, Brinton two to one, Steiner eleven to eight, and Wollmann the ratio about equal.

The disposition to this disease seems also to be greater *in early life*, although in autopsies ulcers are more frequently found in old persons, for the simple reason that in some cases the affection lasts for a very long time before it proves fatal. It is a well-established fact also that in women the ulcer frequently manifests a marked tendency to perforation at about the twentieth year of life.

Individuals of feeble constitution, and those who are chlorotic or anæmic, are more commonly affected than persons of a robust habit of body, although the disease attacks even homines quadrati.[1] So also parturition and nursing may be mentioned as occasional causes, on account of the loss of humors attendant upon these conditions, while the feebleness of the general health in persons inclined to tuberculosis makes them more readily attacked than others (see below, the complications of the ulcer). In all these conditions the common etiological factor is probably an impaired nutrition and abnormal lacerability of the walls of the vessels.

The varying frequency of the disease in different regions shows that the occurrence of gastric ulcer is influenced also by the *climate* and the *mode of life*, although, at present, we know nothing definite upon this point of the etiology.

Of special interest, finally, is the occurrence of gastric ulcer after extensive *burns of the skin*, a lesion which Falk[2] regards as mainly due to the inhibition of the gastric circulation, resulting from the depressed action of the heart. Another interesting fact is the coincidence of gastric ulcer and *trichinosis*, observed by Ebstein and others—the explanation here probably being the production of acute gastritis and hemorrhagic erosion by the trichinæ.

Symptomatology.

Of all the symptoms of gastric ulcer none is so constant as *pain in the stomach*. It is absent only in exceptional cases, and particularly when the affection runs a very rapid course (*e. g.*, No. X. of Abercrombie's cases). The pain varies considerably in character and intensity in different patients, and, of course, possesses no pathognomonic character. More important than the question whether the pain is burning, gnawing, raging, etc., is the *time* when it occurs. This is generally from a few minutes to a quarter of an hour after eating, or sometimes even a whole hour; it is specially apt to occur after the ingestion of coarse indigestible food, and it does not cease until after digestion is completed—that is, until several hours have elapsed, or until the food is discharged by vomiting.

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[1] **Short, thick-set, squarely built individuals.—TRANS.**

[2] **Virchow's Archiv. 53. S. 68.**

The attempt has been made to localize the situation of the ulcer by the time which intervenes between the taking of food and the occurrence of the pain. Thus in ulcers at the cardia the pain is supposed to occur *immediately* after the food is swallowed; but the consideration that after even a few minutes—in Busch's well-known case[1] of duodenal fistula, even after twelve minutes—the food has passed the pylorus, is opposed to such a conclusion, no less than is clinical experience. Pinel, for instance (Abercrombie, p. 36), reports the case of a man with ulcer at the pylorus, who suffered the most violent pain immediately after taking even the smallest quantity of food.

These cardialgic attacks may be excited, not only by errors in diet, but also by exposure to cold, mental excitement, menstruation, severe bodily exertion, etc. The pain is increased by external pressure, tight clothing, and a variety of other causes. This circumstance would be of greater diagnostic importance, if it had not been noticed in some cases that the pain was *relieved* by pressure upon the epigastrium, and by similar manœuvres (for example, see L. Müller, p. 66; Fenwick, p. 329; Abercrombie, p. 46; Henoch, I., p. 19, and others). The pain is sometimes continuous, sometimes *intermitting*. It is probably scarcely possible to account for these intermissions by an explanation which is applicable to all cases. Bamberger's supposition, that the paroxysms "may depend upon an extension of the ulcer, and the implication of new nerve branches," sounds very plausible. That it does not, however, answer for all cases is shown by the fact that even small ulcers may occasion frequent and severe attacks of pain; and that, on the other hand, as Brinton, particularly, insists, the pain in the case of large ulcers is often entirely absent for several days. If we bear in mind that many of the nerve extremities in the base of the ulcer are denuded, and exposed to the action of the acids which are present in the contents of the stomach, to the influence of heat, and to the mechanical irritation caused by solid substances—all of which are clearly "depressing" agents (though at first only for the motor nerves)—we may easily suppose that these agents act like other depressants, and induce first a stage of increased irritability, and then a stage in which the irritability is blunted, or finally completely destroyed. By rest and the neutralization of the chemical depressants the irritability of the nerves may be restored, while an extension of the ulcer may expose new nerve fibres to the injuries above described.

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[1] Virchow's Archiv. 14. S. 167.

Not only the time when the pain is felt, but also the *locality* is of some importance. Usually the pain is said to be situated in the epigastrium, and to be aggravated by pressure in this region. Too much stress, in my opinion, should not be laid upon the diagnostic importance of this symptom. Aside from the fact that by far the greater number of ulcers are situated upon the posterior wall, and therefore cannot be directly affected by pressure on the epigastrium, it should also be borne in mind that in the neighborhood of the ensiform cartilage deep pressure produces an unpleasant painful sensation even in healthy persons, probably on account of the compression of the anterior gastric plexus. A better guide, therefore, for the diagnosis of the existence and situation of a gastric ulcer is the observation that the *pain is concentrated in the umbilical region* (corresponding to the greater curvature), *or in the dorsal region*, near the first lumbar vertebra and the lower portion of the dorsal vertebræ (corresponding to the lesser curvature and posterior wall of the stomach), to which latter localization Cruveilhier particularly has called attention ("point rachidien"). Still another criterion of the situation of the ulcer is afforded by the fact (Osborne, Brinton), that *in many, though of course by no means in all cases, the position of the patient has a very important influence upon the production and mitigation of the attacks of pain.* Thus when the ulcer is upon the posterior surface of the stomach, the patient lies bent forwards, in order to avoid the paroxysms of pain, lies upon his right side when the ulcer is upon the cardia, etc.

Traube has met with some interesting cases, in which, instead of the usual pains in the stomach, or at the same time with them, *oppression in the chest* occurred, which he explained as a "radiation neuralgia," induced by the transmission of the irritation of the gastric nerves to the pulmonary filaments of the vagus. Similar radiations were noticed by Traube in the form of neuralgia of the intercostal nerves, or of the left brachial plexus, and by Brinton in the form of neuralgia in the right shoulder; the neuralgia in this latter case having, moreover, a tangible anatomical cause in an adhesion of the stomach to the surface of the liver (see Luschka, Neck, p. 409. Relation of the Posterior Supra-clavicular Nerves to the Phrenic Nerve). In a case seen by L. Müller the attacks finally came to be attended by very active sympathetic sensations in the arms and legs, terminating in convulsive movements of the extremities.

Vomiting is absent in very few cases. In one hundred and twenty cases, collected by L. Müller, the symptom was entirely absent, or did not appear until the end of the disease, in only one-fourth of the cases. It is interesting to notice that in these exceptional instances the situation of the ulcer was unusual; thus the duodenum was affected in nine, the lesser curvature in six, the anterior wall in seven; while the posterior wall, the pylorus and the cardia were affected in only one each. Almost always the vomiting is accompanied by pains in the stomach—the explanation evidently being that the emesis and the cardialgia are both excited by the irritation of the gastric nerves. Usually pain is felt in the stomach for some time before the first attack of vomiting occurs; so also as a rule the cardialgia is the first symptom, and when it has reached its height then the vomiting takes place. As soon as the vomiting occurs, the cardialgia disappears. This circumstance, to which there are of course some exceptions, points to a common origin for both symptoms. Moreover, the fact that the vomiting and pain usually continue until the stomach is completely emptied, shows that these symptoms are generally due to an excessive excitation of the gastric nerves by the contents of the stomach. The patient soon learns for himself that his attacks are very apt to be induced by coarse indigestible food. In rare cases vomiting occurs with an empty stomach, probably because the saliva, which is swallowed, excites a secretion of acid in the neighborhood of the ulcer, the secretion overflowing the surface of the ulcer, and thereby producing an energetic reaction on the part of the exposed ends of the nerves.

As the pain may arise from other causes than errors in diet, so also the vomiting may occasionally be excited by mental disturbances, bodily exertion, etc.

The *vomit* contains food in the different stages of digestion, sarcinæ, bile, etc.,—in short, nothing which is at all characteristic of gastric ulcer. The occurrence of large quantities of blood in the matters vomited—the *hæmatemesis*—is a much more important sign, and, with certain limitations, is indicative of this lesion.

Hæmatemesis is, upon the whole, much rarer than the simple vomiting, which, as above observed, occurs in about four-fifths of the cases; while, according to L. Müller's compilation of one hundred and twenty cases, vomiting of blood occurred in only thirty-five, or not quite one-third of the cases. It is also much less common than ordinary vomiting as a symptom of gastric diseases in general; while, on the other hand, none of the causes of bleeding from the stomach give rise to such frequent and severe hemorrhages as ulcer of the stomach. The most copious hemorrhages naturally occur from erosion of a large vessel, a symptom which can occur only when the ulcer, not necessarily a large one, has perforated all the tissues of the stomach. The symptoms of hæmatemesis vary with the quantity of blood poured out into the stomach. The smaller hemorrhages, which almost necessarily occur when the ulceration is spreading, often escape notice, because the effused blood does not induce vomiting, but passes off in the evacuations, which are scarcely ever examined unless there is some special reason for so doing. In other cases the stomach, in consequence of the long-continued habit of vomiting, is intolerant of even a small quantity of blood, which is therefore rejected, and gives to the vomited matters a dirty brown color from conversion of the hæmoglobin into hæmatin under the action of the gastric juice. These minor hemorrhages do not materially disturb the general health of the patient; the case is different, however, when more copious hemorrhages take place from the erosion of vessels of considerable size. The occurrence of this more serious complication is usually announced by increased pain, anxiety, an attack of faintness, and nausea; a sensation of sickness at the stomach occurs, and large quantities of pure blood are vomited, followed by profound syncope, or death when the amount of blood rejected is very large. In copious hemorrhage a portion of the blood always escapes by the bowels as a blackish substance, and in some cases all the extravasated blood passes off in this way in the form of tarry fæces. This result is most apt to occur when the hemorrhage is gradual, or is not so copious as to distend the stomach, and excite vomiting by mechanical irritation. Finally, in some rare cases of profuse hemorrhage there is no discharge of blood externally, either by the mouth or rectum: the patient becomes suddenly faint and pale, cold at the extremities, convulsed, and within a few moments a corpse. The autopsy in such instances shows in the stomach enormous masses of blood, which may entirely fill and form a cast of the organ.

The hæmatemesis very generally returns, either because the obstructing clot is dissolved by the gastric juice, or is forced out by the movements of the stomach, or because a fresh rupture is produced by the degeneration of the wall of the vessel (atheroma, etc.: see above). This accident is favored by everything which favors congestion of the stomach, particularly by over-indulgence in food, which not only acts as a mechanical irritant, but also excites a more copious secretion of gastric acids. Whatever the explanation, the hemorrhage is very generally observed to occur soon after eating.

Dyspepsia, or "difficult digestion," as shown by a lack of or perversion of the appetite, by increased thirst, unpleasant taste in the mouth, weight in the epigastrium, and eructation of gases and acid fluids, is a very common, but not constant, accompaniment of gastric ulcer. This train of symptoms will naturally always occur whenever the normal digestion of food is for any reason disturbed—a condition which exists in most of the cases of gastric ulcer.

There is nothing in the pathological anatomy of the ulcerative process itself which necessarily involves the simultaneous occurrence of a gastric catarrh. In fact, in *acute* ulceration the mucous membrane appears perfectly normal, while the perforating ulcer is directly characterized by an *absence* of any inflammatory proliferation. Usually, however, sooner or later, the ulcerative process becomes complicated with a *chronic gastritis.* This results in the occurrence of derangements of digestion. Perhaps also another cause for the latter is to be found in the ulcer itself. The solution of continuity in the muscular fibres, which takes place in deep ulcerations, involves an interruption of the work performed by the muscles, and this interruption will be still more likely to occur when the stomach has become adherent to adjacent organs. Such a loss of muscular power may naturally result in an imperfect expulsion of the chyme from the stomach, in an insufficient impregnation of the food with gastric juice, a less complete formation of peptones and absorption of the same by the wall of the stomach —in brief, in a delay of the entire process of digestion, with decomposition of the ingesta. In fact, these symptoms of deranged digestion are very commonly found in cases of gastric ulceration ; sometimes also there is very marked pyrosis, with salivation and distention of the epigastrium, which add greatly to the distress of the patient.

Notwithstanding these digestive derangements, the *nutrition* is by no means always impaired ; indeed the appearance is sometimes that of perfect health, and it is only too familiar an experience to see death occur suddenly from hæmatemesis or perforation in a patient who has hitherto been entirely free from symptoms of ill health, except slight dyspepsia of long standing, and has been able to attend to business.

The explanation of these startling cases probably lies in the fact that the gastritis accompanying the ulceration may be very limited, and that the disease, with its depressing factors—pain, vomiting, and hemorrhages—shows intervals of quiet, in which the ulcer begins to heal, but only sooner or later to relapse. When, on the other hand, the symptoms of dyspepsia continue uninterruptedly for years, accompanied by cardialgia, vomiting, and hemorrhages, the patient finally becomes more and more cachectic, loses flesh and strength, becomes œdematous about the ankles, and sometimes succumbs to the disease from simple exhaustion.

Almost invariably gastric ulcer is accompanied by obstinate *constipation.* We have seen above that in all probability the normal movements of the stomach are impeded in this affection. Furthermore, Traube[1] and Radziejewski[2] have shown that between the movements of the stomach and those of the intestines there exists a certain reflex sympathy ; and this fact, it seems to me, serves to explain the sluggishness of the bowels so often noticed in cases of gastric ulcer. The occurrence of constipation is favored also by the relaxation of the abdominal muscles produced by the continued vomiting, and, above all, by the circumstance that, in consequence of the vomiting, but little of the ingesta remains to be converted ultimately into fæces.

In a disease which usually lasts for months or years, which at every meal reminds the patient of the hopelessness of his condition, which is constantly exhausting his strength, causing him violent pain and disturbing his rest, it is but natural that the patient should gradually lose courage and fall into a state of profound *depression of spirits.*

Another symptom, upon which the older writers laid much stress, is *amenorrhœa.* There are, however, so many depressing forces at work during the course of ulceration of the stomach, which are likely to produce this symptom, and amenorrhœa is so common an occurrence under other circumstances, that it is far from being a characteristic of the disease.

Besides the symptoms above described, which belong to the disease directly, others occasionally occur which are to be regarded as *complications.* These require a brief consideration.

The most important, and certainly the most disastrous event in the course of gastric ulcer is the *perforation.* Sometimes this is the first indication of the existence of the hitherto latent disease ; the patient, who has previously made no complaint, except of slight dyspeptic symptoms, is suddenly attacked by severe pain in the epigastrium, rapidly spreading over the whole abdomen, and followed by the other symptoms of peritonitis from perforation—meteorism, nausea, etc. Under these circumstances death almost always occurs within two or three days, with symptoms of deepening collapse. When adhesions have previously been formed between the stomach and adjacent organs, the course of the case is somewhat more favorable, the perforation here resulting in the formation of a saccular cavity, in the establishment of fistulæ, etc. (see above, Pathological Anatomy). Usually, however, the saccular collections of pus and ichor burst after a while into the peritoneal cavity, and induce a fatal result. In other cases of antecedent adhesion, pneumothorax may be produced by rupture through the diaphragm, or, when the perforation occurs inferiorly, a fistula may be established between the colon and stomach, resulting in stercoraceous vomiting (see Case XI. in Abercrombie, p. 55). In very rare cases the perforation takes place externally, with the formation of a simple gastric fistula ; van Swieten has published some remarkable instances of this kind. Occasionally abscesses occur in the liver and spleen as a result of pylephlebitis, starting in a gastric ulcer (Bamberger and others).

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[1] Gesammelte Beiträge, etc. II. S. 354.

[2] Dubois-Reichert's Archiv. 1870. S. 1. et seq.

After preparations for the perforation have been progressing for some time, its final occurrence seems to be favored by certain circumstances, such as meals (by inducing excessive flatulent distention or energetic movements of the stomach), bodily exertion, direct mechanical injuries to the stomach, etc.

Perforation is not an infrequent termination of gastric ulcer, since, according to Brinton's estimate, one out of every seven or eight cases ends in this way. There is a striking difference in regard to the average age at which this accident occurs in the two sexes, the average age in women being twenty-seven, and in men forty-two years.

Another very important complication which deserves mention is *tuberculosis*, because there has been supposed to be a certain causal relation between gastric ulcer and this disease, similar to that which exists between cancer and tuberculosis, the intermediate factor in both cases being the regressive metamorphosis produced by the primary affection. Dittrich (Papellier), in one hundred and three autopsies with *non-cicatrized* ulcers of the stomach, observed phthisis and tuberculosis eighteen times ; Steiner (Virchow) in one hundred and ten cases of ulcer—both cicatrized and open—pulmonary phthisis, thirty-three times ; Wollmann, however, in forty-eight cases only five times.

Whether in these cases there really is a causal connection, such as accords with our present conceptions of tuberculosis, or whether the connection is merely an accidental coincidence of different morbid processes, must at present remain undetermined; to decide the point, larger statistics are necessary, which bear directly on the question whether the pulmonary processes are caseous or purely tuberculous. In the article on Cancer of the Stomach we shall consider this subject at greater length.

The *course* of the disease is *chronic* (ulcus "chronicum"); sometimes, however, it is apparently acute, as, for instance, when, after a latent stage of uncertain duration, intense symptoms of ulceration, or perhaps perforation, suddenly occur. Experiments have demonstrated the possibility of a very acute occurrence of the ulcer, but, as a rule, the completion of the perforation requires several weeks; on the other hand, cases have been known in which the disease lasted only from twenty to thirty days. As has been already mentioned, intermissions and relapses are quite common.

That there are cases which from the beginning to the end run a *latent* course is proved by the fact that cicatrized gastric ulcers are found after death in persons who have never suffered from any gastric affection during life. These cases show, at all events, the entire *curability* of the disease—a fact, moreover, which has been placed beyond doubt by the results of treatment. But, besides these cases of complete recovery, we constantly meet with others in which, although, anatomically, the cure seems to be perfect, yet, clinically, we encounter sequelæ which are frequently of a serious character.

For instance, persistent *cardialgia* sometimes results from the lacerations to which some of the gastric nerves are subjected, either by the contraction of the cicatrix, or by the adhesions which have formed between the cicatrix of the ulcer and the adjacent abdominal organs. The cardialgia is particularly apt to be excited when these adhesions are made tense by the active movements of the stomach in digestion, and under such circumstances even vomiting may occur. The influence of this impediment to the movements of the stomach manifests itself, not only in attacks of cardialgia, but also in the various forms of *dys-*

pepsia. The occurrence of the latter is favored also by various other conditions connected with the cicatrization of the ulcer, such as the loss of a portion of the secreting surface of the mucous membrane, and of a portion of the capillary lymphatic absorbents; furthermore, by the consecutive gastritis, which is itself due to the stases produced by the constringing action of the cicatricial tissue upon the blood-vessels.

According to the situation of the cicatrix, the cavity of the stomach may present various forms of contraction and of subsequent dilatation. The dilatation of the stomach is especially apt to occur in connection with cicatricial stricture of the pyloric region; while, on the other hand, narrowing of the entire lumen of the stomach may result when the cicatrix is situated at the cardia.

In the latter class of cases the final result is *death.* Hemorrhage and perforation are the two accidents which suddenly induce a fatal termination, while in another class of cases the patient falls into a gradual decline, becomes extremely reduced by the cardialgic attacks, vomiting, and digestive disturbances, and finally succumbs to some complication, such as pneumonia, diarrhœa, etc. When tuberculosis occurs in connection with gastric ulcer, the pulmonary affection will naturally contribute its share to the cachexia and fatal result. The same remark will apply to other complications, especially those affecting the circulatory apparatus.

Diagnosis.

Every physician of experience will concede the correctness of my statement when I say that, although the diagnosis is sometimes perfectly clear, yet in *numerous* cases certainty in the diagnosis is entirely out of the question. I refer particularly to the cases of *sickly chlorotic girls or women, with menstrual disorders, who complain of cardialgia and indigestion, and in whom tenderness of the epigastrium is found on physical examination.* These are the cases which, at least for myself, have most frequently presented difficulties in diagnosis. Sometimes, to be sure, the simultaneous existence of neuralgia or hysterical complaints—globus, clavus, etc.—and the absence of any connection between taking food and the vomiting, afford some support for the exclusion of ulcer; still, in this class of persons gastric ulcer is so frequently attended by nervous symptoms that the presence of the latter does not contra-indicate the existence of ulcer. Of late years I have been accustomed in these doubtful cases to begin with a course of treatment as if gastric ulcer were actually present, such as the enforcement of strict abstinence from all food, except beef broths, etc. (see below). If the disease were not considerably benefited by this diet, I excluded the existence of an open ulcer, and treated the case with hydrochloric acid, iron, electricity, etc., as if it were one of anæmic dyspepsia. My success with this method has been very gratifying, and by its aid I have finally cured women who had previously been treated for years in vain.

In other cases the diagnosis may fluctuate between ulcer and *gastralgia* (purely nervous cardialgia). Usually, however, the occurrence of pain after eating, the continuous character of the pain, and its aggravation by external pressure; the absence of other neuralgias, of uterine disease, of hysteria, or of hypochondria; the coëxistence of dyspepsia and the frequency of the vomiting, point rather to ulcer than to nervous cardialgia. Still, it must be evident from the symptomatology of gastric ulcer, as considered above, how erroneous it would be to regard these differential indications as pathognomonic. Moreover, we shall presently see in the article on Gastralgia that in certain cases a positive discrimination between the two diseases is absolutely impossible. Under such circumstances the use of electricity has sometimes aided me in the diagnosis. If the pain disappear within a few minutes after the application of a constant current from a battery of from twenty to forty cells, I regard this fact as indicative of cardialgia; in gastric ulcer I have *never yet* been able to produce a cessation of the pain by the use of the constant current. It is scarcely to be expected that this aid to differential diagnosis will be infallible; in fact, I am satisfied that many gastric neuralgias resist the action of the current just as constantly as other neuralgias, and I therefore do not attach any value to the results of the application of electricity, except when they are of a positive character, and particularly when the pains are relieved by electricity during digestion. More numerous trials are necessary to confirm the correctness of this test.[1]

Still more difficult in some cases is the *differential diagnosis between ulcer and cancer of the stomach.* So long as no tumor can be felt in the epigastrium, and no development of carcinoma can be discovered in other organs—such a discovery almost invariably indicating cancer of the stomach—the diagnosis can usually be only a probable one. In doubtful cases the following may be regarded as general indications in favor of ulcer as opposed to cancer: youthful age, healthy appearance, slight emaciation after a long continuance of the disease, strikingly marked variations in the condition of the patient, and very copious hæmatemesis.

Only in very rare cases is it possible to confound the cardialgic attacks, which accompany gastric ulcer, with *hepatic colic.* In the latter affection the pain is situated rather in the right hypochondrium than in the epigastrium; the gall bladder can frequently be felt; the liver is enlarged; icterus is generally present, and the digestion during the painless intervals is undisturbed. In certain cases of hepatic colic the vomiting may, to be sure, become continuous, and when this occurs in connection with severe epigastric pains, the diagnosis may for a time be uncertain until the doubt is removed by the escape of biliary concretions in the evacuations.

Finally, the question not unfrequently arises whether the case is one of gastric ulcer or of *chronic gastritis.* The difficulty of deciding this question is the greater because a part of the symptoms in cases of gastric ulcer are not uncommonly due to a coexisting inflammation of the mucous membrane. Here the history of the case, the fact that the disease has originated independently of definite external influences, the absence of hæmatemesis, of the complete intermissions, and of the characteristic paroxysms of pain, must decide in favor of gastric catarrh, in which, on the other hand, the symptoms are more persistent, the pain is not so severe and is less circumscribed, and the quantity of blood in the vomited matter, when there is any at all, is at all events very insignificant.

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[1] For the history of a case of this kind, see the article on Gastralgia.

Fortunately, notwithstanding all these difficulties, there is still a class of cases in which the diagnosis of gastric ulcer can be made with the necessary certainty. In other cases, however, difficulty may be encountered in the fact that the complex of symptoms mentioned in the symptomatology is not present in its entirety. In fact, numerous instances have been reported in which cardialgia, dyspepsia, hemorrhage, and vomiting have each been the only symptom of a gastric ulcer whose existence was demonstrated by the autopsy. As I have already mentioned, it is occasionally advisable, for the purpose of diagnosis in doubtful cases, to treat the patient as if he were really suffering from gastric ulcer, and then to base the diagnosis upon the success or failure of the treatment. This practice is the more justifiable, because, in the morbid conditions which are liable to be mistaken for gastric ulcer, the stringent diet and other measures suited to the treatment of gastric ulcer can at all events do no harm.

Prognosis.

When we consider that cicatrices are met with at autopsies nearly as frequently as open ulcers, and that of the latter many might have healed had time permitted, one is tempted to take a favorable view of the prognosis. Still it is to be remembered particularly, that the milder, spontaneously cicatrizing ulcers do not come under the observation of the physician during life, and that, consequently, when the ulcer can be diagnosticated with certainty, the presumption is that it is of large size, or that it has penetrated to a considerable depth. This presumption becomes a certainty, when serious *hemorrhages* occur, because, as already stated, this accident can happen only when all the coats of the stomach have been perforated as far down as the peritoneum. Severe hemorrhages are always an alarming symptom ; in one hundred and twenty cases observed by L. Müller, hemorrhages occurred thirty-five times, and proved fatal in fourteen ; in Steiner's one hundred and ten cases, fatal hemorrhage occurred in seven.

Hæmatemesis is dangerous, moreover, on account of the exhaustion which it produces, and on account of the tendency to relapse. The extreme danger of *perforation* and the unfavorable significance of such complications as pulmonary phthisis and disease of the heart or vessels require no further consideration. Severe persistent vomiting and long-continued tormenting cardialgia ultimately produce exhaustion, and thus impair the prospect of recovery.

But even in those cases in which we have reason to suppose that the ulcer is cicatrizing, the prognosis is not absolutely favorable, because relapses are not infrequent, and the cicatrices may give rise to permanent alterations of the lumen of the stomach and to obstinate cardialgia and dyspepsia.

Nevertheless the treatment of gastric ulcer is by no means a thankless task, since by far the greater number of cases are cured, if we include those patients who are relieved for a long time of all their complaints, but subsequently have a relapse.

Treatment.

It must be evident, from the conjectural nature of our knowledge of the causes of gastric ulcer, as shown in the chapter on etiology, that a rational *indicatio causalis* is out of the question. There are certain points of prophylaxis, however, which should always receive attention; for instance, in chlorotic or sickly persons, or others, who are supposed to be more or less predisposed to gastric ulcer, it is necessary to insist upon the avoidance of strong irritants to the mucous membrane of the stomach, especially when the organ is empty; to prevent the too long continuance of violent acts of vomiting, and in regions where gastric ulcer is very prevalent, to alter, as far as possible, the usually very perverted habits of life in the lower classes, at least among those who are especially predisposed to the disease, etc.

Indicatio morbi.—As there is no prophylactic, so there is no specific for gastric ulcer. Still much may be accomplished by treatment, if we keep in view what is the main object of all rational treatment, viz., the necessity of placing the diseased organ under such conditions that all causes which interrupt the curative process are as far as possible eliminated.

Aside from all theories in regard to the origination and cicatrization of gastric ulcer, we must regard as settled this fact, that *an ulcerated surface will not be prevented from healing unless it is kept irritated by external causes.* This fundamental law of treatment is, however, always violated, unless we make it our first rule to allow *no solid food*, or at least none which cannot by mastication be converted into a soft, pulpy mass. It was upon this principle that Cruveilhier, the author of the views at present entertained in regard to gastric ulcer, recommended the use of *milk*, which for years has been the sovereign article of diet in this affection, and the efficacy of which has been demonstrated in thousands of cases. Not only does this article of food contain in itself the principal nutritive ingredients—albumen, fat, carbo-hydrates, and salts—but it also possesses the special advantage in the treatment of gastric ulcer, that the soft caseous coagula formed in the stomach are less irritating to the ulcerated surface than are other substances, such as hard-boiled eggs, pieces of meat, bread, cabbage, potato, etc., all of which quit the stomach quite unchanged, while the casein of the milk, by the time it has reached the upper part of the small intestine, has become finely comminuted. In some patients, unfortunately, the milk coagulates in the stomach in the form of large tough lumps, and then its further use must be discontinued. Another article of diet, which is even less likely than milk to inflict mechanical injury upon the ulcerated surface, is the *solution of beef*. This forms a soft mass resembling an emulsion, which glides over the wall of the stomach as a fine slime without exciting any irritation. With the exception of these two articles of food, and perhaps some soup containing the white of egg, or barley water, nothing else should be given, at least in the beginning of the treatment. After a while, when the principal symptoms have disappeared, and there is reason to suppose that the ulcer has begun to cicatrize, we may resort to the light diet previously described. The following articles of food are to be particularly avoided, on account of the mechanical injury which they inflict upon the wall of the stomach: all *leguminous vege-*

tables, *fruits*, and *brown bread*, the latter being more or less injurious to the diseased mucous membrane according to the amount of bran which it contains; also *oatmeal gruel*, which is specially recommended by some physicians, but which is liable to cause considerable discomfort to the patient, because some of the spiculated oat-grains may pierce the surface of the ulcer, and thus excite severe irritation. Potatoes also should be prohibited, except in the form of a soft soup made with milk, on account of the danger of swallowing small spiculated fragments.

Still more baneful than mechanical injuries to the open surface of the ulcer, is the action of *chemical* irritants. As we have seen above, it is unquestionably to the acids present in the contents of the stomach that the origination and extension of the ulcerative process are ultimately due. Partly with this view and partly for the purpose of relieving the pyrosis and intensely acid vomiting, modes of treatment have been suggested which are supposed to counteract the formation of abnormal quantities of acid. Thus the addition, to the milk, of lime water (a third part), or of magnesia, has been recommended to neutralize the contents of the stomach. But aside from the fact that we have no certain guide as to the quantity of alkali necessary for neutralization, and that, according to physiological observation, an excess of acid merely serves to excite an energetic secretion of acid, the benefit, even if the correct quantity of alkali were known, would be a very questionable one. The temporary *neutral* reaction would directly promote the acid fermentation, especially the conversion of lactic acid into butyric acid, carbonic acid, and hydrogen, and would delay the escape of the food through the pylorus, because normally this does not occur until the contents of the stomach have become more intensely acid.

In this way, therefore—that is, by the use of remedies directed against the formation of acids—we can scarcely expect to accomplish our object. The prospect of success will be *more encouraging if we endeavor to prevent as far as possible a long-continued collection of acids in the stomach.*

This indication may be carried out in various ways.

The most direct method of emptying the stomach is naturally by means of the stomach-pump. The reports of Schliep, who has quite recently used this mode of treatment, sound very encouraging, several of his patients having been relieved and cured by the use of the pump only two or three times. Still I doubt whether the instrument will speedily come into general use for this purpose, because its employment presupposes a considerable degree of toleration on the part of the patient, and because, on the other hand, physicians will frequently be deterred from its use by the well-founded fear of bringing the tube in direct contact with the affected part, and thus possibly producing perforation or hemorrhage.

Another method, which is much more frequently used and which is likewise very efficacious for temporarily emptying the stomach, is the use of alkaline-saline mineral waters or their salts. The well-marked efficacy of these waters depends upon the happy combination of three active salts, the chloride of sodium, the carbonate of soda, and the sulphate of soda. That the chief effect is due to the latter salt is shown beyond doubt by the favorable experience we have daily had in the treatment of gastric ulcers with the artificial Carlsbad salt, which consists almost entirely of sulphate of soda (Ziemssen). The derangement of appetite, and other dyspeptic symptoms, which are noticed when Glauber's salt is used alone, do not occur with the use of the Carlsbad salt, but seem to be prevented by the presence of the chloride of sodium and carbonate of soda. The beneficial effect of the alkaline-saline mineral water may be at least partly explained also by the well-known influence of common salt in promoting digestion and by the action of the carbonate of soda in dissolving mucus and diminishing the excessive acidity of the contents of the stomach; but the most important result of the remedy is undoubtedly the expulsion of the ingesta from the stomach into the small intestine, an effect which is due chiefly to the sulphate of soda.

The correctness of this view is shown also by the fact that the water from simply alkaline-muriatic springs is of no decided benefit in gastric ulcer, so long as it is not taken mixed with the salt of an alkaline-saline spring. Moreover, experience teaches that mineral waters of a warm temperature are more efficacious and probably less irritating to the stomach than the cold waters, and that none of the alkaline-saline springs are equal to the Carlsbad. The Tarasp, Marienbad, Rohitsch, and other similar waters, should therefore be heated to about 104° F. The Tarasp water, on account of its containing larger quantities of free carbonic acid and chloride of sodium, seems to be more apt to produce hemorrhage than the other mineral waters belonging to this class, among which may be mentioned also the Bertrich (90° F.), the Elster, and the Franzenbad, the two latter being distinguished by their containing iron. If, instead of a course of the water at one of the springs mentioned, the natural or artificial Carlsbad salt be used at home, the best method of using the latter, in my experience, is that described by Ziemssen: one table-spoonful of the salt is dissolved in a pint of luke-warm water; of this the patient drinks, fasting, about a fourth part, and repeats this quantity every ten minutes, so as to be about three-quarters of an hour in taking the whole amount. Then the patient is to wait half an hour longer before he takes his breakfast, which is usually followed by one or two watery discharges. If he has more than two, or none at all, the quantity of salts taken the next day must be regulated accordingly, but the amount of water in which the salt is dissolved is to remain the same (one pint).

The possibility of thus relieving the stomach of its irritating ingesta, temporarily, naturally suggests the desirability of maintaining the beneficial effects of keeping the ulcer free from the corroding action of the gastric juice for as long a time as possible. This object can be accomplished most certainly, as was previously suggested, by allowing the stomach entire rest for a considerable time. The entire feasibility of such a plan is shown by the fact, demonstrated both by physiological experiment and by clinical trials, that a considerable quantity of nitrogen can be introduced into the body by means of the *meat-pancreas injections*[1] recommended by me, and that a person can be nourished in this way *per rectum* for a long time without suffering any sensation of hunger. This annoying mode of administering nourishment is, however, generally unnecessary. The same good results may be obtained by using the meat solution[1] devised by Rosenthal and myself. The beneficial effects of this treatment are especially evident in patients who cannot bear even the "easily digestible" food.

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[1] Deutsches Archiv für klinische Medecin. X. 1 et seq. The recipe for making this injection is as follows: take about five ounces of finely scraped meat, chop it still finer, add to it one and a half ounces of finely chopped pancreas free from fat, then add about three ounces of luke-warm water, and stir to the consistence of a thick pulp. This is given as an enema, care having been taken to wash out the rectum with water about an hour before.

I recently treated a patient with gastric ulcer at the Jena clinique, who, after taking the meat solution a short time, became so disgusted with it that she refused absolutely to continue it. A milk diet was substituted, but without any improvement in the symptoms. The failure of this substitute, and the brilliant success of the meat solution in another patient near by, induced her now to follow up the use of the meat solution. After a very short time the symptoms of the ulcer disappeared, and the patient was discharged cured.

The reason why the meat solution is borne so well by patients with ulcer lies, not only in the resemblance of the preparation to an emulsion, as already mentioned, but also in the slight demand which this article of food makes upon the functional activity of the stomach. In the preparation of the solution the meat is reduced by the slight amount of heat and the treatment with acid to much the same condition as during digestion in the stomach; and as far as we can judge from the apparent change which the meat has undergone, and from the results which are obtained in the relief of the dyspeptic symptoms, it is reasonable to suppose that the preparation is directly absorbed by the stomach or passes unchanged into the small intestine. Under such circumstances the diseased stomach naturally finds rest, and the ulcerated surface, freed from the incessant irritation, is placed in a condition favorable to cicatrization. For the past two years I have treated all well-marked cases of gastric ulcer with the meat solution, and have uniformly met with good results. After a few days the pain and vomiting usually cease, and the healing advances so rapidly that after two or three weeks the patient may return with impunity to a more solid diet. Of course caution is still necessary, and the convalescent's stomach should not be taxed with the digestion of any food that is not easily digestible. Below I give in brief a few illustrations of the treatment of gastric ulcer with the meat solution.

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[1] Sammlung klinischer Vorträge von Volkmann. No. 62. (See also p. 474.)

Miss V., sixteen years of age, has suffered for eighteen months with cardialgia, vomiting, and eructations. One year ago hæmatemesis occurred; for the past six months all the symptoms have been aggravated; cardialgia occurring regularly fifteen minutes after each meal; vomiting is now less frequent. Anorexia; no relief to the pain by change of position; constipation; pallor of the mucous membranes; pressure on the epigastrium painful; motion excites eructations. Ord.: Carlsbad salts, one pot of beef solution, one quart of milk daily, soup, and rusk. After eight days of this treatment the pain disappeared entirely. After eighteen days, during which the food was retained by the stomach, and the meat solution was always taken with relish, chicken and other easily digestible articles of diet were resumed. Gradually a more substantial diet was permitted, which was borne without any inconvenience. Discharged cured.

H. H., thirty-four years of age, has suffered from gastric derangement since his twenty-second year. Weight in the epigastrium, especially after a hearty meal, for the past three years, with vomiting, eructations, and blood by stool. Epigastrium tender on pressure. Ord.: solut. carnis, and poultices. Decided improvement by the sixth day. The solution was taken with relish.

E., forty-three years of age, has suffered from gastric derangements and vomiting for more than three years. On one occasion great prostration with severe epigastric pain, which compelled her to keep in bed for a week, and was attended by great thirst and giddiness. One year ago a copious hæmatemesis; for the past three weeks again she has been confined to her bed. Continual eructations and black evacuations. The patient is obliged to maintain the prone position, on account of the severe pains which occur when she lies on her back or right side. The abdomen is unusually resistant, and pressure on the region of the pylorus painful. Ord.: solut. carnis, etc. After five days the pains decreased, and in three days more were entirely gone. On change of diet the severe pain recurred after eating a hard-boiled egg, and the patient was obliged to resume the solution, under the use of which she gradually improved, and left the hospital, having gained ten pounds in weight.

M., thirty-one years of age. A dyspeptic for four years; cardialgia, eructations after eating, vomiting with occasional admixtures of blood, and on one occasion copious hæmatemesis with passage of blood by stool. For a year past severe cardialgic attacks immediately after eating, especially after taking indigestible food. Eructations, loss of appetite, vomiting of food frequently with small quantities of blood, constipation. Pain on pressure in the epigastrium, and in the lower part of the back on the left side, near the spine. Ord.: solut. carnis, etc. After ten days, pressure in the epigastrium less painful; in the third week the sensitiveness near the spine was entirely gone, and that in the epigastrium considerably diminished. Appetite improved. By the beginning of the fourth week the usual diet was gradually resumed, but after five or six days the pain in the epigastrium returned, and the meat solution was therefore again resorted to for three weeks longer, at the expiration of which time ordinary food was again given, and was well borne, so that two weeks later the patient was discharged apparently perfectly cured.

My method of treating gastric ulcer is briefly as follows:

The patient is confined to his bed during the course of treatment; hot poultices are applied to the abdomen (at night a Priessnitz compress, or ice, if hemorrhage be threatened), and active movements of the body are to be avoided as much as possible. During the first few days Carlsbad salt (one tablespoonful in a pint of lukewarm water) is given in the morning. If the salt fail to properly evacuate the stomach, the stomach-pump may be cautiously used, and the gastric surface washed with lukewarm water. The diet of the patient consists, at the start, of one pot of the beef solution per diem, corresponding to half a pound of beef, and to this I usually add, for breakfast and dinner, some milk and a few pieces of rusk, which should not be swallowed until they have become thoroughly softened and masticated. The beef solution is taken pure, or it may be stirred in bouillon with a little of Liebig's extract of meat, and a little salt added or not, as desired. All the food should have a lukewarm temperature. After from two to three weeks I place the patient upon a light diet, consisting of pigeon, chicken, purée of potatoes, thicker soups, wheat bread, etc.; and after eight days longer I gradually return to coarser food.

Since I have adhered strictly to this method of treatment, I have never found it necessary to resort to any other remedies, not even to the use of morphine to relieve the cardialgia, which with this regimen begins to disappear, like the other symptoms, in the course of a few days. Still less have I been obliged to use the other remedies, so highly praised in former times, *nitrate of silver*, *subnitrate of bismuth*, etc.

The *subnitrate of bismuth* was originally recommended for cardialgia (Odier, 1786), in doses of from two to twelve grains, four times daily, fifteen minutes before eating. Subsequently the remedy was given in much smaller doses; more recently it has been recommended again, especially by the French physicians, in larger quantities, even up to the enormous amount of four drachms a day, and has gradually acquired the reputation of a specific. The fact, however, that the bismuth is usually given in connection with morphine and a regulated diet, and that its efficacy is praised in the most minute as well as in the largest doses, is enough to cast serious doubts upon its supposed virtues. For those who are disposed to try the remedy, because they regard its efficacy as proved by the circumstance that, notwithstanding the impossibility of explaining its action, it has for a century maintained its position among the remedies for diseases of the stomach, and especially for gastric ulcer, I append the usual dose: five to eight grains with or without one-twelfth of a grain of muriate of morphine three times daily.

Another metallic salt, which has been particularly praised in the treatment of this affection, is *nitrate of silver.* Like bismuth, it was first introduced for the relief of cardialgia (Autenrieth, Jun., 1829; and Johnson). At first it was supposed to act by its influence upon the nervous system, but of late, in view of the improbability of this explanation, it has been thought to act rather by virtue of its caustic properties, as in the case of relaxed ulcers on other parts of the body. But if we look at the matter closely, it is hardly probable that such a small amount of the drug as is usually given, a pill containing one-sixth of a grain, will seek out from the whole surface of the stomach the very spot of the ulcer; and even if it did, it is absolutely incredible that the pill, after having become coated in the stomach with a layer of chloride of silver, can exert any alterative effect upon the condition of the ulcer. Nor even when the salt is given in solution (one-fourth grain to the ounce), and upon an empty stomach, is it any easier to see how the tablespoonful of fluid, should it fortunately reach the surface of the ulcer, is to have any appreciable effect.

I am convinced that the more strictly the above directions in regard to diet are carried out, the smaller will be the list of remedies in use for the treatment of gastric ulcer. The same remark will apply also to the other remedies which are used for the relief of certain prominent symptoms, and which will now be briefly considered.

For the relief of the severe *pain* in gastric ulcer the best remedy is *opium* or *morphine*, the latter either hypodermically or in cherry-laurel water (two and a half grains to the ounce; dose, twenty drops). Gerhardt regards the *muriated tincture of iron*, three or four drops in a wine-glassful of water, several times a day, as better for this purpose than any narcotic, and as, moreover, less disturbing to digestion, because it does not precipitate the peptones. When the pain and tenderness are limited to a small spot on the anterior abdominal wall, so that there is reason to infer the localization of the ulcer at that point, or the existence of a local peritonitis of the anterior wall of the stomach, the continuous application of a light ice-bag is especially to be recommended. If the strength of the patient permit, a few leeches may also be applied. In cases where the epigastrium is much swollen, and the pain is suspected to be partly due to flatulent distention of the stomach, if the use of charcoal prove ineffectual, the attempt may be made to remove the gas by means of the stomach-pump. Mustard plasters, blisters, the actual cautery, the moxa, etc., to the epigastrium, have sometimes given relief, but they can probably be dispensed with in all cases.

Nothing relieves the obstinate vomiting so effectually as the strict observance of an unirritating diet. If this do not succeed, the best remedy is morphine, given as above described. In its failure benefit is occasionally derived from swallowing small pieces of ice. Others recommend creosote, half a drop or a drop in one or two tablespoonfuls of water at a dose; tincture of iodine, three drops in a mucilaginous vehicle, etc.—remedies which have no better support than a poverty-stricken empiricism. While direct treatment is rarely required for simple vomiting, the occurrence of *hæmatemesis* renders the employment of special measures indispensable. Absolute rest should be ordered, abstinence from food, ice pills, a bladder of ice to the epigastrium, and mustard-plasters to the feet. In case these fail, hypodermic injections of ergotine may be tried (injection fluid: ergotine fifteen grains, distilled water two drachms, alcohol forty minims; dose, fifteen drops). For other particulars I refer to the article on Gastric Hemorrhage. For *pyrosis* antacids may be used, with the restriction previously given. In other respects the dyspeptic symptoms require nothing more than the faithful observance of dietetic rules. This, together with the occasional use of the Carlsbad salt, will generally suffice also to remove the disturbances arising from constipation. When constipation still remains after the ulcer has healed, a course of treatment at Franzenbad or Elster may be prescribed. These waters, aside from their cathartic properties, are particularly adapted to promote convalescence on account of their ferruginous ingredients. The rhubarb, aloes, and other pills, which are ordinarily used for "habitual constipation," should be avoided as far as possible; but if they are necessary, pills made of gamboge, or the compound rhubarb pill (from one to three at bed-time) are to be preferred, as they may be given for a long time without harm. If iron be also given as a restorative, its acknowledged tendency, when long continued, to derange digestion should be borne in mind, and only those preparations which are least injurious in this respect should be chosen, such as the lactate, or pyrophosphate of iron, etc. In chlorotic patients the administration of iron is indispensable, because the chlorosis is directly connected with the origination and relapses of the gastric ulcer. When *perforation* occurs—that most disastrous event in the course of gastric ulcer—the only treatment in most cases is to induce euthanasia. Energetic measures are however not to be neglected, in view of the fact that recovery occasionally occurs under these circumstances (Ross, Schliep), apparently because the stomach was empty at the time of the perforation. These measures consist in the administration of large doses of opium (one grain every three hours), morphine injections, absolute rest, abstinence from all food, fomentations, or the ice-cold compresses, which are preferred in Germany, and the excellent effect of which in peritonitis I can myself confirm. If the vomiting still continue, it would be proper, in order to prevent the further escape of the ingesta through the perforation, to resort to the cautious use of the stomach-tube, as has been successfully done by Schliep.

Even after the ulcer has entirely healed, many patients require prolonged treatment for the various sequelæ which are produced by the cicatrization and by the adhesion of the stomach to other organs: cardialgia, defective secretion of gastric juice, diminished peristalsis, or, in the worst event, stricture at the cardia, or pyloric stricture with dilatation of the stomach. Each case will require a special mode of treatment, according to the predominance of one or other of these sequelæ. Most of the latter, particularly the results of strictures, together with their treatment, are considered more fully in other articles. In all of them the first and most important object is a careful regulation of the diet, so as to avoid taxing the powers of the stomach. The patient is, therefore, to be kept upon a light, easily digestible diet for some time after the beginning of convalescence. It should never be forgotten, also, that relapses in this disease are not infrequent, and that for this reason caution in the use of food is imperative even after complete recovery.

Tumors of the Stomach.

A great variety of tumors occur in the wall of the stomach: *fibroids*, *myomata*,[1] *lipomata*, *papillomata*, *sarcomata*, *carcinomata*, *tubercle*, and *adenomata*.

Of all these neoplasms, carcinoma is the only one which possesses any clinical interest, because in the case of some of them the growth is too small to be felt, while others, like sarcoma, are not to be distinguished by palpation from carcinomatous tumors. The symptoms produced by these neoplasms are only to a very small extent directly dependent upon the tumor; they are to be regarded rather as the expression of the complicating catarrh, or as the results of the accidental situation of the growth, for instance, the dilatation of the stomach produced by pyloric stenosis when the tumor occupies this region. Moreover, all the above-mentioned tumors of the stomach have hitherto been noticed so rarely in comparison with carcinoma that a clinical description of them is entirely out of the question.

How cautious we should be, in the case of a gastric tumor, against inferring that the affection is something else than carcinoma, even when the particular circumstances of the case almost compel us to deviate from the usual diagnosis of cancer of the stomach, is shown by the following instance observed at the Jena clinique:

M., a school-teacher, twenty-five years of age, ill for sixteen months with eructations, constricting pains in the epigastrium, loss of appetite, and emaciation. The examination gave the following result: cachectic appearance, excessive emaciation, muscular system moderately developed. The left half of the epigastrium is slightly swollen, more in the upright than in the recumbent posture. Under the left hypochondrium a tumor appeared during inspiration, resembling the spleen in position, form, and consistence, and towards the middle line of the abdomen joining another tumor with irregular borders, which also moved with the respiration. The latter tumor extended a finger's breadth below the umbilicus, and to the right joined the liver, whose edge could not be felt. Percussion note dull-tympanitic. At the left of the umbilicus was *a cutaneous tumor half the size of a pigeon's egg, of a bluish red color, firm, painless, circumscribed, and having a somewhat mammillated appearance upon its surface ;* this tumor, the patient stated, had made its appearance six months before. *On the skin of the chest and abdomen were small hard tumors, from the size of a pea to that of a bean,* projecting slightly above the level of the skin, and of the same color as the surrounding parts; in the groin there was also a small nodule of a bluish color.

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[1] A full description of this interesting, and not very rare form of tumor is given by Virchow (Onkologie. III. 126). According to the direction of the growth, he divides myoma of the stomach into an internal and an external variety.

The œsophageal tube was introduced for the purpose of diagnosis. Near the cardia an obstruction was encountered, which, however, could be easily overcome. The larger red bluish tumor near the umbilicus was cut out by two elliptical incisions, and sent to the Institute of Pathological Anatomy for microscopical examination.

The *structure of the cutaneous tumor* presented throughout a connective-tissue character, being composed of a network of fibrillar connective-tissue rings filled with groups of quite large cells, some of which were round, others polygonal, lying in a delicate sharply outlined reticulum. Here and there were interspersed extensive, irregularly circumscribed, groups of large fat cells. The sweat glands were strikingly enlarged, and their excretory ducts unusually convoluted. According, therefore, to the results of the microscopical examination, it seemed probable, if not certain, that the tumor belonged to the class of *connective-tissue neoplasms.*

The vomited matters contained food and mucus, but never blood or other abnormal ingredients. The emaciation continued to progress, the cutaneous nodes and the abdominal tumor increased in size, and death ensued at the end of three months.

At the *autopsy* no enlargement of the liver was found ; the entire upper wall of the stomach, from the cardia to the greater curvature, was occupied by a large tumor which was adherent to the slightly enlarged and otherwise normal spleen. The glandulæ epiploicæ along the smaller curvature were considerably enlarged, fatty, and adherent to the stomach by means of dense connective tissue. Towards the beginning of the antrum pylori the rough and uneven mucous membrane became gradually smooth, and lost its tumefied appearance. In the middle of the tumor was a large, round, deep perforation, which apparently originated from an old ulcer; in this neighborhood the swelling of the mucous membrane was greatest. The wall of the stomach was considerably thickened; even after hardening in absolute alcohol, it still measured one and a half centimetres in thickness. All the layers of the wall of the stomach were involved in the neoplasm. The muscular coat had a reticulated appearance, and was hypertrophied ; the serous coat uniformly white and studded with superficial nodules. No adhesion was found between the diaphragm and the stomach. The microscopical examination of the tumor by Prof. W. Müller showed that the case was undoubtedly one of fibrous *carcinoma* of the stomach, with a marked development of connective tissue. Cancerous elements were found also in the lymphatic vessels of the glandulæ gastro-epiploicæ.

If the rare gastric sarcoma, of which Virchow has recorded a remarkable example in his work on tumors (II., S. 352), can ever be diagnosticated during life, this certainly seemed to be an instance of it. The tumor occurred in a young man; it had never induced vomiting of blood or substances like coffee grounds, so that there was no reason to suppose that there was any ulceration of the surface of the stomach; the tumor extended uniformly over the greater portion of the organ, and finally—the most important point in the diagnosis—multiple fibro-sarcomata were present in the skin of the body, which were apparently of a metastatic character. The autopsy, however, showed that the gastric affection had been due to a simple carcinoma ventriculi, and that the microscopical structure of the cutaneous tumors, notwithstanding their apparently unmistakable metastatic nature, afforded no indication as to the real character of the gastric tumor.

It is possible, that at a later examination of the cutaneous nodules their epithelial character might have been better developed, and the case thus have become clearer. As it was, the examination was made at a time when they had already been growing for half a year. The fact, therefore, that we were left completely in the lurch by the most objective factor in the diagnosis—the existence of numerous nodular tumors situated where they were directly accessible to examination—makes the diagnosis of sarcoma ventriculi during life a mere matter of guesswork.

Essentially, therefore, the clinical aspects of tumors of the stomach coincide with those of *gastric carcinoma.*

Cancer of the Stomach. Carcinoma Ventriculi.

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Onkologie. I.—*Waldeyer's* Arbeiten über den Krebs. Virchow's Archiv. 41. S. 470; Virchow's Archiv. 55. S. 67; *Volkmann's* Sammlung klinischer Vorträge. No. 33.—*Acker*, Zur Pathogenese der Geschwulstmetastasen. Deutsches Archiv f. klin. Med. XI. S. 171.—The text-books of *Bamberger*, *Henoch*, *Siebert*, and others, on diseases of the abdomen.—*W. Brinton*, Diseases of the stomach, etc. 1859.—*Wilson Fox*, The diseases of the stomach. 1872.—*Martius*, Die Combinationsverhältnisse des Krebses und der Tuberkulose. Diss. Inaugur. Erlangen. 1853. (*Dittrich.*)—*Lebert*, Traité prat. des maladies cancéreuses, etc. Paris. 1851. —*Oppolzer*, Carcinoma ventriculi. Klin. Vortrag. Wiener medic. Wochenschr. 1865. 1-3.—*Kussmaul*, Ueber die Behandlung der Magenerweiterung und eine neue Methode mittelst der Magenpumpe. Deutsches Archiv f. klin. Medic. VI. S. 455.—*Luton*, Magenkrebs. Nouveau diction. de médecine, etc. Publié par Jaccoud, Paris, 1871, with a copious bibliography of French literature.—*Friedreich*, Ein Fall von Magenkrebs. Berliner klin. Wochenschr. 1874. No. 1. —*F. Riegel*, Ueber die therapeutische Anwendung der Cundurangorinde. Berliner klin. Wochenschrift. 1874. No. 35.

Pathological Anatomy.

The theory of the development of carcinoma has recently undergone such a radical transformation, more especially through the epoch-making writings of Waldeyer, that I shall not hesitate, in the following brief consideration of the pathological anatomy of gastric cancer, to make a free use of the convincing expositions and conclusions of this investigator.

According to Waldeyer, carcinoma is an "atypical epithelial neoplasm." In the stomach the neoplasm always originates in the cylindrical epithelia of the glands of the mucous membrane (Cornil). These cells proliferate *in a downward direction*, and the growth is most active after they have pierced the muscularis mucosæ, and have reached the loose submucosa. Here the "cancer nodules" are first formed. The heaps of short cylindrical epithelial cells ("cancer bodies") lie in a stroma formed from the connective-tissue elements of the mucous membrane. In this stroma, and in the immediate neighborhood of the cancer-bodies, a small-cell infiltration, containing numerous newly-formed vessels, makes its appearance, which presses apart the enlarged glandular sacs, and, superiorly, separates them from the upper surface of the mucous membrane. (See the instructive illustrations in Waldeyer's articles. Virchow's Archiv. 41, plate 11.

Fig. 3; and 51, plate 8, fig. 12.) According now to the special forms of development which are taken by the stroma, or by the cancer-bodies, we obtain different varieties of gastric cancer, which differ from each other essentially in their external appearance and their microscopical structure. They may all be included under three general forms:

1. Carcinoma fibrosum (scirrhus), fibrous cancer.
2. Carcinoma medullare, medullary cancer.
3. Carcinoma colloides (c. alveolare), gelatinous cancer.

While *the first two of these varieties are distinguished from each other by the different degrees of development of the stroma* —in fibrous cancer, the connective-tissue stroma predominating unmistakably over the cancer-bodies, and in medullary cancer the cancer-cells predominating over the stroma—the third variety, *colloid cancer, is characterized by a metamorphosis, which affects the cancer-bodies themselves,—the colloid degeneration of the cancer-cells.* The different varieties of cancer may be described more in detail, as follows:

1. *Carcinoma fibrosum.*—Fibrous cancer appears either as an annular, dense, callous infiltration, or in the form of circumscribed nodes. "In marked cases the walls of the stomach, with perhaps the exception of the fundus, appears to be converted into a rigid mass, an inch or more in thickness, which frequently presents nodular protuberances directed, generally, towards the interior of the organ. The coats of the stomach are no longer distinguishable, and, since the mucous membrane is replaced by the morbid structure, the latter lies bare upon its internal surface. The cavity of the stomach is also narrowed by the encroachment of the tumor" (Rokitansky). The neoplasm burrows into the muscularis in the form of long bands, in the neighborhood of which the muscular fibres at first undergo hypertrophy, but afterwards become atrophied as the cancer-bands grow more luxuriantly. The mucous membrane and peritoneum are not involved in the cancerous degeneration until a later period. The microscopical examination shows an excessive development of connective tissue with interspersed roundish or oblong nests of cells.

2. *Carcinoma medullare.*—Medullary cancer is composed of

circumscribed fungous nodes, or of annular protuberances, which extend over large portions of the wall of the stomach, and soften upon their surfaces into ulcers. The ulcerated surface is covered with ragged tufts, which wave to-and-fro when immersed in water. Microscopically, the heaps of cancer cells appear, on the whole, to predominate over the masses of connective tissue; but it is quite common to find, also, at some points the texture of fibrous cancer. The ulceration is produced by fatty degeneration of the stroma and cancer cells, aided by the destructive action of the gastric juice.

3. *Carcinoma colloides.*—Colloid cancer, which is usually disseminated diffusely, is characterized by uncommonly large alveoli, in which, instead of the cancer cells, a gelatinous mucous substance is found, resulting from a peculiar metamorphosis of the cancer cells, called the colloid metamorphosis. The ulceration advances slowly, and the disease may attain an enormous extent, involving even the omentum and other portions of the peritoneum. This form of cancer is much rarer than the two former, occurring in only from two to eight per cent. of the cases of gastric cancer. The other varieties—for example, villous cancer—may without difficulty be classified under the three preceding general forms.

In the great majority of cases the cancerous tumor is *situated* at the *pylorus*. Out of three hundred and sixty cases analyzed by Brinton, the pylorus was affected in sixty per cent., the cardia in ten per cent. Luton's analysis of Lebert's, Dittrich's, and Louis's one hundred and two cases showed the tumor to be situated:

At the pylorus	in	59 cases.
On the lesser curvature	“	17 “
At the cardia	“	8 “
On the anterior and posterior surfaces	“	5 “
On the greater curvature	“	2 “
Over the whole stomach	“	1 case.
At different points	“	7 cases.

In the majority of cases the stomach is the only organ affected; in other instances the cancer extends to the peritoneum, lymphatic glands, liver, colon, pancreas, etc.; while in still other cases the stomach is involved secondarily by a cancer, which has originated in other organs.

When primary carcinoma of the stomach extends to other organs, it involves most frequently the liver, and in particular cases the disease develops in the latter to a much more marked degree than in the stomach itself. In one hundred and sixty cases of gastric cancer examined at the Pathological Institution in Prague, Dittrich found secondary cancer of the liver forty-three times; of the peritoneum, twenty-two times; of the lungs, nine times; of the retro-peritoneal glands, five times; of the rectum, twice; and of the ovaries, ribs, bladder, and seminal vesicles, once each. In Brinton's compilation, also, the highest ratio was presented by the liver, viz., one hundred and five times out of four hundred and thirty-one cases. These reports, therefore, give an important clinical result, viz., *that in about one-fourth of the cases of gastric cancer, the liver also exhibits carcinomatous degeneration.* We shall recur to this circumstance in the symptomatology, where we shall also have to speak of the perforations into various organs, resulting from the extension of the cancerous ulceration to the external surface of the gastric wall.

Among the *anatomical resultant conditions* which are brought about by the development of the carcinoma in the stomach itself, the first in importance is the change in the position of the organ. Usually when the pylorus is affected, it is adherent to the surrounding parts; in rare instances, however, this fixation does not take place, and then the pyloric portion, in consequence of its increased weight, sinks into the lower regions of the abdomen (sometimes even as far as the symphysis), where it may be felt as a freely movable resistant tumor; or it may become adherent to the uterus, intestines, etc. Besides being changed in position, the stomach also undergoes alterations in its lumen—dilatation or stenosis, according as the neoplasm is located at the pylorus or at the cardia—or local constrictions (hour-glass shape). The muscular coat of the stomach is sometimes hypertrophied, and sometimes, as the disease progresses, atrophy takes place; the mucous membrane presents, to a greater or less extent, the signs of chronic inflammation.

Among the *complications* of gastric cancer — pneumonia, œdema of the lungs, pleuritis, thrombosis of the veins, etc.—special mention should be made of *tuberculosis*, the causation of which in this connection may possibly require some other explanation than the marasmus produced by the cancer. To this point, however, we shall devote a fuller consideration in the chapter on symptomatology. Finally, the combination of carcinoma with the *round ulcer of the stomach* is particularly interesting, not only from a pathologico-anatomical, but also from an etiological point of view. In one hundred and sixty cases, Dittrich found this combination eight times; in some of the cases cicatrices of healed ulcers existed unconnected with the cancer; in others the carcinoma appeared to be implanted upon an old ulcer. Waldeyer speaks of quite superficial nodules, with an indurated, slightly granular, ulcerated base, which were with difficulty to be distinguished from simple round ulcers, even with the aid of the microscope, and he comes to the conclusion that there is no pathologico-anatomical reason why "a process, which at first is simply ulcerative, may not in time be converted into a cancerous one, if the epithelial elements of the wall of the stomach, instead of passively disintegrating, take part in the proliferation of the border of the ulcer." This subject brings us to another chapter, to the etiology of the disease.

Etiology.

No organ of the body is so frequently the seat of cancer as the stomach. According to Tanchou's compilation of 9,118 fatal cases of malignant cancerous tumors, the uterus was affected more frequently than the stomach by about seven per cent.; but the statistics of Marc d'Espine and Virchow (see Onkologie I. 82), which are probably more accurate, give the greater frequency to the stomach.

Thus in the compilation of the former writer, including 889 cases, the stomach was affected 390 times, and the uterus only 139 times; while Virchow found the stomach affected in 34.9 per cent., and the uterus in only 18.5 per cent.

The average age of persons dying of cancer of the stomach, according to Brinton's analysis of 600 cases, was fifty years. Three-fourths of the patients were between forty and seventy years of age, and two-sevenths—the largest subdivision—between fifty and sixty. If the numbers of persons dying of cancer of the stomach be compared with the numbers of persons alive at the different decades, it will be found that the relatively greatest disposition to the disease occurs from the age of sixty to seventy years. Under the age of thirty the disease is comparatively rare; but one instance is recorded by Wilkinson in which it existed at the time of birth.

Sex appears to exert no decided influence upon the occurrence of the disease; at least this fact is strikingly shown in the reports of those writers who have larger statistics at command. Of the 1,303 cases, collected by Wilson Fox, 680 were males and 623 females.

We are accordingly compelled to look for the causes of cancer in general, and of cancer of the stomach in particular, in the advanced age of the patient, and in the structure, position, and function of the stomach; but how these factors act is at present a matter of mere hypothesis. As above mentioned, the existence of an ulcer or of a cicatrix from previous ulceration seems to produce a certain predisposition to the formation of carcinoma. For a long time a predisposing influence has been ascribed also to chronic gastritis, which Boerhaave (Aphor. § 956) thought occasionally terminated in scirrhus. In fact, it is undeniable that cases of gastritis are observed, which finally end after several years in cancer. Interesting in this connection is the fact noticed by Waldeyer, that "in the most recently formed zones of cancer the tissue was almost as vascular, and filled with heaps of white blood-corpuscles, as it is in inflamed tissue." It is easy to see, therefore, how the exuberant nutrition in inflammation and the relaxation of the connective tissue might favor a rapid growth of the epithelial cells.

The foregoing constitute all the positive facts we possess upon the obscure subject of the etiology of gastric cancer. They are insufficient, however, in my opinion, to warrant at present the expression of decided views in regard to the origin of the disease, however tempting it may be to magnify the results of severe gastric irritation, particularly at the narrow orifices, or of an increased richness of the epithelia in protoplasm into proliferative processes and development of cancer.

The other supposed causes of gastric cancer scarcely deserve mention, *e. g.*, errors in diet, brandy drinking, anxiety, deprivation, and injuries to the epigastrium. The influence of *heredity*, however, seems to be better supported by facts, as, for instance, by the case of Napoleon's family, which is usually paraded as an illustration. In such instances we are obliged to suppose that a certain imperfection in the structure of the mucous membrane is inherited, which in the presence of a determining cause, such as advanced age, etc., ultimately gives rise to an atypical proliferation of the gland sacs.

Symptomatology.

Patients with gastric cancer present the well-known fawn-yellow color, the languor and bodily weakness, the slight œdema, the emaciation, and the other signs of cachexia, which we are accustomed to see in invalids with cancer, and the cause of which probably lies in the absorption of disintegrated products, and in the incessant waste of bodily substance kept up at the seat of the neoplasm. In gastric cancer, however, there is a special cause for the general deterioration of the body, viz., the deranged digestion produced by the destruction, to a greater or less extent, of the glandular structures.

The *hydrops* cachecticus, which is usually limited to a swelling about the ankles, occasionally becomes so excessive as to induce not only general anasarca, but even copious effusions into the pleural cavities, the pericardium, and particularly the peritoneal cavity. In such cases the diagnosis may present great difficulties, as is shown by the following instance, which occurred in the Jena Clinique.

Patient sixty years of age; has general anasarca, which has developed within the past three months. The bowels have been regular, appetite good, and, with the exception of eructations, no digestive derangements of any kind. In addition to enormous ascites, the patient has had hydrothorax and tracheal œdema, and, as a result of the latter, attacks of suffocation. The whole appearance resembled strikingly that of a patient with renal disease, but the urine was always free from albumen. The examination of the heart, lungs, liver, and spleen showed nothing abnormal; palpation in the epigastrium negative, on account of the excessive ascites. The patient died from asphyxia.

The *diagnosis* under these circumstances was a purely symptomatic one: "general dropsy from hydræmia."

At the *autopsy* a *cancer of the pylorus*, as large as a fist, was found, with two secondary nodules of inconsiderable size in the liver.

Another symptom, of still more significance than the mere cachexia, is *hypertrophy of the peripheral lymphatic glands*, especially in the supra-clavicular region (Virchow). Still this sign is often absent, and when present may naturally be due to other causes.

The same may be said of the *pain* and *gastric disturbances*. *Pain* in the epigastrium is absent only in very rare cases, as in the case just mentioned; according to Brinton it is absent in eight per cent. of the cases. In many patients, however, it is a very marked symptom; but in this, as in most other diseases, it is never pathognomonic, notwithstanding all that is said of the specific "lancinating" character of the pain of cancer.

The *gastric disturbances*, on the other hand, which accompany cancer of the stomach, are of greater symptomatological importance, because they point directly to the situation of the disease, and because their entire absence is extremely exceptional, although, as in the above case, they may play a very subordinate rôle. These dyspeptic symptoms have usually been ascribed to chronic gastric catarrh, which is supposed to be a regular accompaniment of the cancer. Probably, however, the coexistence of catarrh is more frequently theoretically inferred than actually present, although it is not to be denied that the constant irritation, which the growing tumor excites in the surrounding parts, may induce and maintain a chronic catarrh of the stomach. Still the symptoms in question are susceptible of another explanation, viz., the diminished secretion of gastric juice, the loss of absorptive power, and the interference with the regularity of the movements of the organ—conditions which of themselves are sufficient to prevent the normal and to initiate abnormal changes in the ingesta.

Among the most constant of the gastric symptoms produced by the carcinoma is *loss of appetite*, being present, according to Brinton, in eighty-five per cent. of the cases. It is a symptom, however, of little value, because, as Brinton points out, it frequently does not make its appearance until late in the disease—a fact which accords with the view expressed above.

Very nearly as frequent as the anorexia is the occurrence of *vomiting*. Brinton, who has made the largest collection of cases, and who has analyzed the individual symptoms statistically, found, in his compilation of one hundred and sixty-seven cases, that emesis occurred most frequently in cancer of the pylorus, and most rarely in cancer of the posterior wall of the stomach; between these extremes came, in descending ratio, cancer of the cardia, of the greater curvature, of the lesser curvature, and, finally, of the middle portion of the stomach. This compilation shows conclusively that vomiting is most frequent when the neoplasm is situated at one of the orifices of the stomach, evidently for the reason that they are both—at all events, the cardia—immediately concerned in the act of vomiting. As this subject has been fully considered above, we remark here only that, when the vomiting occurs in connection with the presence of a pyloric tumor of inconsiderable size, the symptom may be explained by the *irritation* of the pyloric musculature by the tumor and the masses of food; such an irritation resulting in a firm and long-continued constriction of the orifice. The escape of the ingesta into the duodenum is therefore delayed; the retained chyme decomposes, irritates the mucous membrane, and excites vomiting. At a later period, when the pylorus is converted into a rigid, *contracted* orifice, the neoplasm itself interferes with the escape of the chyme from the stomach. When the carcinoma is situated at the cardia, the main factor in the vomiting is the abnormal relaxation of the orifice produced by the irritation of the tumor.

In a very large proportion of cases (about forty-two per cent., according to Brinton), the *vomited matters* contain *blood*, either in a pure, or, far more frequently, in a decomposed form. In the latter case the well-known transformation of the hæmatin, under the action of the gastric acids, gives the discharge the appearance of chocolate or of coffee-grounds. These hemorrhages may arise either from the erosion of small or large vessels, or from vascular congestions induced by the growing tumor, the result in either case being due to the influence which the neoplasm exerts upon the wall of the stomach.

The simplest method of determining whether the color of the discharge is due to *blood*, or to an accidental black coloration of the ingesta, is a spectroscopical examination, which, if hæmatin be present, will show the usual absorption bands between C and D.

In addition to the blood, there will also be found in the vomited matters portions of the food, sarcinæ, mucus, and occasionally *cancerous* particles. The difficulty of finding the latter, and the fact that, after their detachment, they are subjected to the action of the gastric juice, fully account for their rare recognition under the microscope. When, however, the carcinoma is situated at the cardia, the passage of the stomach tube may succeed in bringing away cancerous masses, which have become engaged in the fenestra of the tube, and then a more satisfactory microscopical examination may be made.

The character of the *stools* is not at all constant; in some cases constipation, in others diarrhœa is present.

As regards *temperature*, Brinton is of the opinion that *fever* is a not infrequent symptom of gastric cancer; but this statement requires further confirmation.

More important, in a diagnostic point of view, than any of these symptoms, with the rare exception of the detection of cancerous particles by microscopical examination, is the discovery of a *tumor* in the epigastrium. It needs, however, but a glance at the normal relations of the stomach to the anterior wall of the abdomen to see that the whole of the smaller curvature, the whole of the posterior wall, the cardia, and the pylorus, are not in contact with the abdominal wall, and are therefore not accessible to palpation, and that consequently the presence of a tumor can be expected in only a portion of the cases. When these conditions change, and the tumor—as in the case, particularly of pyloric cancer—becomes lowered in position by reason of its weight, the tumor can be more readily appreciated; still my own experience has been that the cases in which palpation has failed have been very numerous. The position of the tumor naturally varies according to the situation of the neoplasm, and, moreover, according to the degree of distention of the organ, because during digestion the stomach, as is well known, revolves upon its longitudinal axis. This fact is of special value in determining the location of a tumor situated at the pylorus, because, after eating, the tumor is said to usually disappear under the left lobe of the liver; in rarer instances the tumor does not become distinctly palpable until the stomach has been considerably distended.

Brinton has furthermore called attention to the fact that in pyloric cancer the position of the tumor is singularly different in the two sexes. He found that in *men* two-thirds of the tumors lay *above* the horizontal line, which separates the epigastrium from the umbilical region; while in women one-third were situated above, and two-thirds below this line; the majority of the tumors in women, therefore, being situated in the umbilical region. This striking fact is chiefly due, in his opinion, to the use of corsets, which compress the narrow epigastrium, and force the pyloric tumor downwards. If further experience should show that this difference in regard to the sexes is not accidental, the explanation given by Brinton would be a very plausible one.

On *palpation*, the tumor is usually found to present a hard consistence and an uneven surface. Sometimes a distinct pulsation may be felt, due to the movements of the aorta, which are readily conveyed through the solid mass to the abdominal wall; still this symptom has no diagnostic value, because epigastric pulsation is due to a great variety of causes, as probably every one has learned by personal clinical experience.

When the existence of a solid tumor has once been satisfactorily determined by palpation, the first point to be determined in regard to it is its *mobility*. Aside from the previously described movements of the tumor connected with the distention of the stomach, it must, in general, be borne in mind that, as distinguished from other abdominal tumors, gastric tumors are *immovable*. The relations of the stomach to the diaphragm would naturally lead us to expect that a tumor situated in the wall of the stomach would necessarily descend with the contraction of the diaphragm ; but practically this is not the case. On the contrary, cancerous tumors remain in their original position undisturbed by the respiratory movements, probably because the motion imparted to the diaphragmatic surface of the wall of the stomach is readily dissipated laterally in an organ filled with air and fluid. This lateral dissipation of force does not occur to such a marked extent when the cancerous tumor is immediately adjacent to the diaphragm, and extends far enough downwards to be distinctly felt, nor when the tumor is so extensive that the infiltrated wall is comparatively incompressible ; under such circumstances the lower border of the tumor will necessarily follow the excursions of the diaphragm in the same manner as do the liver and spleen.

The following case,[1] which was observed at the Jena Medical Clinique, is an instructive illustration of the *mobility* of a carcinoma ventriculi.

Patient twenty-five years of age ; much emaciated, and a sufferer from digestive disturbances. On standing, the epigastrium was more protuberant than on lying down. Palpation revealed an abdominal tumor, whose hard nodular border extended from the left hypochondrium diagonally across to the level of the umbilicus, and in the right mammillary line could be sharply defined from the lower border of the liver, the transverse diameter of which, across from the right axillary line, was normal. *On inspiration, the lower border of the tumor descended to a very considerable distance—about six centimetres !* Percussion over the tumor gave a tympanitic sound. The œsophageal tube, which was introduced for diagnostic purposes, encountered near the cardia an obstruction, which gave the impression of a membrane projecting into the cavity of the œsophagus. The obstruction was overcome without much difficulty, and after this the further introduction was easily effected.

Notwithstanding this marked mobility of the tumor, a *diagnosis* of neoplasm of the stomach was made, as the symptoms pointed unmistakably in this direction. Still the tumor in this case unquestionably extended upon the anterior wall of the stomach from the cardia as far as the greater curvature !

The autopsy completely confirmed the diagnosis ; a large carcinoma occupied the whole extent of the anterior wall of the stomach from the cardia to the greater curvature ; no adhesions between the stomach and diaphragm.

The *percussion note* over the tumor is usually of a dull, tympanitic character. This sign is the best distinction between cancer of the stomach and cancer of the left lobe of the liver, although, of course, there are occasional exceptions to this rule.

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[1] See Berichte der medicinischen Klinik zu Jena, etc. S. 105.

Thus, in a woman of fifty-one years of age, with a small, nodulated tumor descending below the umbilicus, and distinctly movable synchronously with the respiration, I found that the tumor always gave an *absolutely dull percussion note.* The fact that the tumor descended with the inspiration, and was dull on percussion, induced me to diagnosticate the case as one of carcinoma of the liver. At the autopsy, cancerous infiltration of the wall of the stomach was found, but no affection of the liver.

In doubtful cases, therefore, I place no reliance upon the sign unless the results are positive—that is, unless the percussion sound on frequent examination is distinctly tympanitic.

The symptoms thus far considered—*gastric derangements, vomiting, emaciation, loss of strength, epigastric pain, and signs of a distinct tumor*—are modified, not only, as we have seen, by the situation of the carcinoma, but also still more by the occasional occurrence of *complications.*

The narrowing of the orifices of the stomach by the tumor produces an expansion of the portion of the alimentary canal situated above the constricted part; dilatation of the œsophagus when the stenosis is caused by a carcinoma cardiæ; dilatation of the stomach itself, in case of pyloric cancer. These sequelæ will be considered in special articles.

Usually cancer of the stomach is accompanied by *hypertrophy* of the wall of the organ, particularly the muscular coat, as a result of the impediment which the tumor presents to the escape of the ingesta.

A rarer sequel than gastric dilatation is a *narrowing* or diminution in the size of the organ. This result may be produced either by the location of the neoplasm at the cardia, or by the cicatricial contraction which occurs in cases of diffuse scirrhus.

Perforation, resulting from deep ulceration of the gastric wall, gives rise to a great variety of symptoms. This termination is, upon the whole, rare. In 507 cases Brinton met with it only 21 times, or about four per cent. According to the situation of the tumor, the rupture may take place on the superior, inferior, anterior, or posterior surfaces. *Perforation into the transverse colon* seems to be most frequent. In this case stercoraceous vomiting may occur as a result of the escape of fæces from the colon into the stomach, or the still undigested food in the stomach may pass directly into this lower portion of the intestinal canal. In this way the obstinate constipation, which has been produced by the obstruction to the passage of food into the intestine, may be suddenly relieved, as I had occasion to observe, several years ago, in a striking case at Niemeyer's clinique. *Perforation into the small intestine* is much rarer, and the same is true of external perforation through the *abdominal wall;* in the latter case a fistula is formed like that which is established for physiological purposes. Perforation into the *thoracic cavity* will naturally give rise to pneumothorax, pneumonia, pulmonary abscess, etc., sequelæ, whose detailed description would be out of place here. If, however, the rupture be not preceded, as it is in these cases, by a circumscribed peritonitis with adhesion to the opposite peritoneal layer at the point of perforation, the food and air contained in the stomach escape into the peritoneal cavity and excite the symptoms of perforation-peritonitis. The entire group of symptoms which characterize this form of peritonitis is not always present. In certain cases nothing escapes through the perforation except traces of food, and then fatal purulent peritonitis results without the usual signs of displacement of the large abdominal glands. On the other hand, even when the escape of ingesta is considerable in amount, the usual symptoms of peritonitis from perforation may be absent if the rupture take place into a saccular cavity.

Another complication, at times equally dangerous, is the *hemorrhages* which sometimes occur during the course of cancer of the stomach. The trifling hemorrhages, which give to the vomited matters the well-known coffee-ground appearance, are of frequent occurrence, but those which proceed from the erosion of a large artery are rare, scarcely one in a hundred cases. Under all circumstances hemorrhages accelerate the anæmia and decline of the patient, while the latter form of hemorrhage may obviously lead directly to a fatal result.

By the *extension of the neoplasm* per contiguitatem to the adjacent organs, or by the metastasis of the carcinoma to distant parts of the body, the originally simple character of the disease may be essentially modified. Of such extensions the most important is the secondary disease of the *liver.* Not to dwell upon the results of direct compression of certain portions of the liver by the gastric tumor, such as portal obstruction, and icterus from the pressure of the tumor upon the bile ducts ; the liver is also exposed to a direct propagation of the neoplasm from the stomach, particularly when the growth is situated on the greater curvature, the anterior surface, and especially the pyloric portion of the stomach. The occurrence of secondary disease of the liver is, moreover, specially favored by the distribution of the portal vein in its parenchyma. The proliferation of the neoplasm into the veins of the stomach may easily give rise to thrombosis, and, later, to embolism of the cancer elements into the small branches of the vena portæ in the liver, as has been directly demonstrated in several cases (see, *e.g.*, Waldeyer and Acker). In a similar manner, *i.e.*, by a dissemination of cancerous masses through the lymphatics, we may explain the occurrence of secondary cancer in the *peritoneum* and the impaction of the lacteals of the duodenal villi with epithelial cells (Waldeyer). Furthermore, the implication of the veins of the liver in the cancerous degeneration may give rise to emboli, which, being carried into the branches of the pulmonary artery, induce a secondary *cancer of the lungs*, as is shown most beautifully by the third case in Acker's work. As soon as these pulmonary nodules proliferate sufficiently to rupture into the pulmonary veins, secondary disseminations of the epithelial elements into the general circulation may readily occur. Although it has not been possible to demonstrate this mechanical origin of metastatic cancer in all cases, yet the reports of success are sufficiently numerous to imperatively demand in future a careful analysis of every case with reference to this point.

In addition, therefore, to the symptoms of gastric cancer, and complicating its diagnosis, we may obviously have a great variety of other symptoms due to portal obstruction, to pressure upon the bile ducts, or to secondary cancer of the liver, peritoneum, lungs, etc.

Apparently less directly connected with the carcinoma in the stomach is the not very rare complication with *pulmonary tuberculosis.* Martius, who has published the views of Dittrich upon this chapter of pathology, has collected from the latter's autopsical experience no less than eight cases, in which pulmonary phthisis with tubercles was found as a complication of gastric cancer.

Dittrich's explanation of the connection between the two diseases is as follows: "The decline in health and emaciation induced by the cancerous cachexia are accompanied by an increased activity of the process of regressive metamorphosis, which in its turn gives rise to fibrin-dyscrasiæ. Among these there is one which manifests a clearly tuberculous character. The tuberculosis, therefore, is merely a local expression of the general contamination of the blood." The above is, upon the whole, a succinct account of the views at present entertained upon the possibly intimate relation which exists between tuberculosis and cancer. Not only does the slowly increasing marasmus of cancer favor the occurrence of caseous changes in the products of pulmonary inflammation, but miliary tubercles may be directly produced by caseous degeneration in the neoplasm itself and subsequent absorption of caseous matters.

Cancer of the stomach in rare instances appears, moreover, to induce an atheromatous degeneration of the arteries; and possibly also the amyloid degeneration of the kidneys and other cachectic conditions of like character, which occasionally accompany gastric cancer, are causally related to the disease of the stomach.

In most cases the *course* of gastric cancer is of a markedly chronic character. Upon the basis of one hundred and ninety-eight cases, Brinton estimates the minimum duration at one month, the maximum at three years, and the average at about one year. Such calculations, as this writer expressly says, are of but limited value. How far back are we to trace the beginning of the disease? Does it start with the first symptoms of dyspepsia, etc.? But how are we to tell whether these are due to epithelial proliferation or to an antecedent gastritis? Do not many patients entirely ignore slight disturbances of digestion, while others at the very outset anxiously consult a physician? It is impossible, therefore, in my opinion to more than approximate to the total duration of cancer of the stomach.

Such statistics, which are based upon the subjective symptoms, are, it seems to me, a far less reliable guide, for both diagnosis and prognosis, than a compilation would be which dated the disease from the first appearance of a palpable tumor. With compilations of this kind, the average duration of the disease would, at all events, be brought within much narrower limits, and some inference might be drawn as to the nature of the carcinoma from the acuteness or chronicity of the disease. Of course, to make the statistics strictly accurate in even this method, we should have to exclude many cases, in which the tumor had attained a considerable development before it was examined by the physician, unless a reliable account of its first appearance could be obtained from the self-examination of the patient.

The disease usually begins with the simple symptoms of chronic gastric catarrh, and the danger is not recognized until attention is called to it by the emaciation and rapid loss of strength. During the last few days of life the distressing pain and vomiting generally cease, and the patient lies cold and apathetic, as if already dead. In other cases death ensues suddenly from severe hemorrhage, while in still other patients the case terminates with the symptoms of perforation—peritonitis, pneumothorax, or some one of the severe complications already mentioned.

Diagnosis.

The symptoms which are of the most importance in the diagnosis of the disease—the *cachexia*, *pain*, *anorexia*, *vomiting* with or without *hemorrhage*, the *age* of the patient, and, finally, the detection of a *tumor* by means of physical signs—have already been considered in detail, together with the restrictions to their diagnostic value in individual cases. It remains only to mention that the greater the number of these symptoms which occur together in a particular case, the more certain is the diagnosis of a carcinoma of the stomach ; but I wish to again insist that the discovery of a tumor far surpasses in diagnostic importance any and all of the symptoms mentioned. If, therefore, a tumor of the character above described be found in the region of the stomach, in a patient whose general condition does not directly contra-indicate the existence of a gastric disease and deterioration of the general health, a diagnosis of cancer of the stomach may almost always be made with certainty. At the same time caution is always to be recommended. Cicatricial, non-malignant condensations near the pylorus, such as sometimes occur as a result of gastric ulceration, may lead to an erroneous diagnosis, and are occasionally, during certain stages of the disease, not to be distinguished from gastric cancer. The same difficulty may arise in connection with an encysted, solid, nodular peritoneal exudation in this region, as recently happened to me in a case, in which the subsidence of the tumor, under continued poulticing, first revealed to me my mistake. It is scarcely necessary to mention that cancer of the left lobe of the liver may be another source of error. Attention to the condition of the right lobe of the liver, the time and mode of appearance of the jaundice, the constancy of the vomiting, the hæmatemesis, and the results of percussion, afford some protection. An erroneous diagnosis may also be occasioned by tumors of the omentum and transverse colon. In the first of these cases, the disproportionate amount of dropsy, without other symptoms; and in the other case, the character of the stools, must be our chief guides in the differential diagnosis. Finally, the question may arise, whether the tumor does not belong to the *pancreas.* In the case of small tumors, it is possible that the tumor which is felt is not a neoplasm at all, but merely the head of the healthy pancreas. A glaring mistake of this kind, I am free to admit, was made by myself several years ago.

The patient, a man who died of chronic pulmonary tuberculosis, without discoverable condensations in the lungs, presented the symptoms of a chronic disease of the stomach, with incessant vomiting and constantly increasing emaciation. Gradually a distinctly palpable tumor was recognized in the pyloric region, and became daily more palpable, so that there seemed to be no doubt of its increase in size. And yet the autopsy showed that no pyloric cancer was present; that the tumor which was felt was merely the head of a normal pancreas, and that its apparent growth was due simply to the increasing palpability of the part as the emaciation of the abdominal integuments progressed, and the intestines became more and more empty in consequence of the prostration of the digestive functions.

The supervention of any of the above-mentioned *complications* usually serves to render the diagnosis of cancer of the stomach more certain, as, for instance, the occurrence of peritonitis with clearly marked escape of air into the peritoneal cavity, or the formation of a pneumothorax, a pulmonary abscess, etc.

Much more frequently the practical difficulty is to decide, not whether a cancer is situated in the stomach or in some other organ, but whether in an undoubted case of disease of the stomach the affection is of a cancerous or innocuous nature. The doubt usually lies between *chronic gastric catarrh*, *ulcer*, *and carcinoma.* So long as no tumor can be discovered, the existence of carcinoma is questionable; at best the diagnosis of this disease can only be a probable one. (See above.)

In doubtful cases, a youthful age, the duration of the disease for years, the discovery of a definite exciting cause, the absence of cachexia, and a decided improvement in the symptoms, are all indications in favor of simple chronic *gastric catarrh.* Such an improvement, however, should not induce us to give a positive prognosis and diagnosis.

A short time ago I saw a case in which the diagnosis lay between carcinoma and chronic gastric catarrh. From the fact that the patient decidedly improved and gained in weight upon the maintenance of a suitable diet, I supposed that carcinoma ought to be excluded. The patient was discharged from the clinic, but after a year's interval returned with marked ascites and dropsy of the lower extremities. The autopsy, which was made shortly afterwards, revealed a cancer of the stomach. While it did not necessarily follow from this fact that the former diagnosis of chronic gastric catarrh was at the time erroneous, the case showed that a temporary improvement and gain in weight (about five pounds in eight days), in cases where cancer of the stomach is suspected, is at least a fallacious guide.

In chronic gastric catarrh the vomiting is usually less frequent and the pain less severe; but these are distinctions upon which it is not well to lay much stress. A frequent coffee-ground appearance of the vomited matters is more significant in favor of cancer of the stomach, because in this disease an admixture of blood is of more common occurrence than in simple gastric catarrh.

Still more difficult in many cases is the differential diagnosis between cancer of the stomach and *simple gastric ulcer.* A youthful age, copious hæmatemesis, and the fact that the disease has lasted for years, are decided indications of the latter affection. In gastric ulcer the pain is characteristic, occurring in paroxysms, and alternating with intervals which are free from pain; but, as I have before stated, this symptom should never be regarded as pathognomonic. Still more important, as supporting the diagnosis of cancer, is an enlargement of the peripheral lymphatic glands; but, after all, the most valuable sign is the presence of a tumor in the epigastrium. So long as no tumor can be felt (or no cancerous elements are to be detected microscopically in the vomited matters), the diagnosis must be left undecided. Perhaps assistance may be derived also from observing the effects of treatment. If further experience should show that gastric ulcers heal invariably, or are at least rapidly benefited by the use of the meat-solution, and by carrying out the rest of the treatment previously described, this would be the simplest test in cases of doubt, while it would have the further advantage that even in cancer of the stomach it could do no harm.

Of course when a patient with cancer of the stomach dies suddenly from hæmatemesis, or from any other cause, before there has been any cachexia or other clear symptoms of gastric disease, a diagnosis is of course out of the question. Such a case was observed by Laborie[1] in a man who had recovered from acute rheumatism, and died from hemorrhage as he was about to leave the hospital; the autopsy revealed a cancer at the lesser curvature.

Prognosis.

Carcinoma of the stomach is *not* curable. Observations to the contrary, particularly of what appeared to be cicatrices of healed cancer in the stomach, are not convincing, because in the complete absence of cancerous proliferation in the neighborhood of the cicatrix, the former nature of the lesion could scarcely be determined, while a partial cure with disappearance of cancer bodies at certain parts only shows that, notwithstanding a local tendency to cure, the disease has not yet run its entire course. Reports, also, like the recent one of Friedreich, that tangible tumors had disappeared under the use of remedies, will not stand close criticism, unless it can be shown by post-mortem examination that the place of these tumors is occupied by a diffuse layer of cicatricial tissue which has the least possible resemblance to the cicatrix of an ulcer.

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[1] *Bouchut*, Nouveaux Eléments de la pathologie générale. Ed. III. p. 288.

In regard to the rapidity with which the carcinoma runs its fatal course, I have no new data to offer. Serious impairment of digestion, frequent vomiting (and therefore the situation of the carcinoma at the orifices), the repeated loss of large quantities of blood in the vomited matters, a marked cachexia from the start, and the intercurrence of some of the more alarming complications, all tend, in proportion to the extent of their development, to hasten the fatal termination. Conjectures in regard to the probable duration of the disease are always precarious; even when death seems to be imminent, the patient may still live for days in a state of complete apathy and collapse.

Treatment.

It is evident from the preceding remarks that a *radical treatment* is out of the question. Although numerous specifics have been recommended, none of them has gained a lasting reputation. From the hard soap recommended by van Swieten (Comm. in Aphorism. § 957) down to the recently recommended carbolic acid, the whole series merely shows how powerless medical skill has been in all times against this disease. The latest remedy suggested is the cundurango bark:

℞. Cort. Cundurango	℥ ss.
Aquae	℥ xij.
Macera hor. XII	
Dein coq. ad	℥ vj.

Cola. S. Take one or one and a half tablespoonfuls twice daily.

Under the use of this decoction, in a case of Friedreich's, the indurated and tender tumors which were distinctly felt in the epigastrium, together with the glandular enlargements in the left supra-clavicular fossa, disappeared in a striking manner, and the improvement continued at all events for half a year. Time and experience will decide in regard to the value of this remedy. In the single case in which I have tried it I found no benefit from its use; the patient died from perforation of the stomach between one and two months after the beginning of the treatment.

Riegel, who tried it in six cases of cancer of the stomach, came to the conclusion that cundurango exerts no specific action upon the cancerous process; all of these patients died of their disease, without any noteworthy indications of improvement at the autopsy. He regards the drug, however, as a powerful stomachic, calculated to relieve the dyspeptic symptoms.

As regards the use of mineral waters, their power to cause absorption is equally inefficacious.

Since the causes of gastric cancer are unknown, an *indicatio causalis* is out of the question. In view, however, of the possible relation which may exist between carcinoma and chronic inflammations, as pointed out in the chapter on etiology, chronic gastric catarrhs, occurring in old persons, should at all events be energetically combated from the start.

In other respects the treatment is purely *symptomatic.* The two most important indications are *regulation of the diet* and *relief of the pain.*

The object of *regulating the diet* is to control the vomiting, to alleviate the pain so far as it is due to an excessive irritation of the sensitive wall of the stomach by indigestible or fermenting food, and to check the cachexia by proper nourishment. In general the same rules will apply here as were previously given in regard to the selection of easily digestible food. Oppolzer calls particular attention to the fact that in many cases only cold food should be allowed, as warm food excites vomiting. In cancer of the stomach, also, as well as in gastric ulcer, the beef solution deserves a fair trial; still, since the disease is incurable, it should be borne in mind that, whatever dietary regulations are adopted, the inclinations of the patient are to be consulted, and no fixed bill of fare should be forced upon him. Any food, therefore, which he desires, which is not positively injurious, and is not too irritating or entirely indigestible, may be allowed. If these regulations are ineffectual, it is well to give the stomach complete rest for a time by the employment of nourishing injections, especially the injections of meat and pancreas—a plan which is, moreover, absolutely indicated in cases of marked stenosis of the cardia. When the vomiting and the pain have been relieved in this way, the use of easily digestible food by the mouth may be resumed; still it is advisable as a rule to give only very small quantities at a time, and, above all, in a fluid form.

In the treatment of obstinate *vomiting*, besides the regulation of the diet, special remedies may occasionally be required. The most useful of these are narcotics and cold. Among the *narcotics*, opiates, particularly morphine in connection with cherry-laurel water, as in the following prescription:

℞

Morphiæ muriatis	gr. jss.
Aquæ laurocerasi	ʒ v.

M. S. Fifteen to thirty drops in the attack.

The morphine may also be given in the form of hypodermic injection. The latter mode of administration answers also for other narcotics, such as belladonna, etc.

Cold is best employed in the form of cold compresses applied to the epigastrium. The patient may at the same time be allowed to swallow small pieces of ice. Carbonated drinks may also be tried: Seltzer water, champagne, etc. All of these measures are indicated, according to Bamberger, only when the vomiting is not induced by stenosis of the cardia or pylorus, because, when this condition exists, their administration involves the risk of retention of the food, and passive dilatation of the stomach and œsophagus. It is scarcely possible, however, even under such circumstances, to dispense with narcotics, if the patient be exhausted by frequent vomiting; while the danger of dilatation can nowadays be obviated by the use of the stomach-pump.

The second leading indication—the *relief of pain*—likewise requires the bold administration of *narcotics*, especially morphine. Given internally in the form just described, or injected hypodermically in a dose of a sixth of a grain, it will temporarily remove the pain, and give the unfortunate patient the desired night's rest.

Besides these two leading indications, the following, directed to particular morbid symptoms, deserve consideration.

The presence of *blood in the vomited matter* requires no special treatment so long as the quantity is small, and particularly when the hemorrhage manifests itself only in the occasional occurrence of a coffee-ground appearance of the vomited matters. When the hæmatemesis is more copious, ice, astringents, etc., may be employed, in accordance with the directions given more fully in the article on Hemorrhage from the Stomach.

For the relief of *pyrosis* antacids may be tried, and for abnormal *fermentative changes* in the stomach either creosote (half a drop at a dose in pill form or in barley water), or benzine (seven and a half minims at a dose). These remedies will sometimes also relieve the vomiting associated with these conditions. In the case of offensive eructations of putrid matters, Oppolzer and Wilson Fox recommend powdered charcoal.[1]

In cases of dilatation of the stomach from pyloric stenosis, the use of the stomach-pump once a day may be resorted to, in accordance with Kussmaul's suggestion. Where the case has not progressed too far, this measure gives the patient much relief, diminishes the sensation of acidity, fulness, and distress, perceptibly reduces the dilatation, lessens the frequently extraordinary acidity of the contents of the stomach, and reduces the frequency of the hemorrhages from the ulcerated surface, probably because the removal of the acid corrosive substances from the stomach diminishes the destruction going on at the site of the cancer. Finally, the washing of the stomach may also relieve the constipation, probably by removing the hebetation of the overtaxed gastric nerves, which thus again become sensitive to weaker irritations, and able to resume their function of initiating the peristaltic movements of the intestines.

In other cases, in which the use of the stomach-pump is objectionable, and the *constipation* cannot, therefore, be relieved in this secondary manner, injections, irrigations, etc., may be used for this purpose. Sometimes, also, a dangerous meteorism may be arrested by means of purgative clysters, as happened in a striking case of Bamberger's. For the relief of flatulence, Walsh recommends the oil of cajeput in doses of from one to three drops on sugar, or better in pill form.

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[1] **The charcoal may also be administered in the form of Rosenthal's tablets.**

In the dangerous *complications* arising from perforation and other causes, peritonitis, pneumothorax, etc., our main reliance, though but a feeble one, is opium and ice, and in desperate cases paracentesis.

The *dropsy*, which sometimes attains such dimensions that the patient suffers more from the hydrothorax and ascites than from the gastric disease, is best treated by diuretics, such as the acetate of potassa, thirteen grains every two hours. Diaphoresis, as is well known, is of no advantage unless energetically carried out, and this is obviously contra-indicated by the feebleness of the patient. The same remark will apply to the use of violent drastics, which only increase the exhaustion.

In *conditions of collapse* recourse must be had to the usual stimulants, particularly strong wine, the use of which should in general not be denied the patient during the whole course of the disease.

Softening of the Stomach—Gastromalacia.

Hunter, Philosoph. transactions. Vol. 62. p. 447; the main facts given also in: *M. Baillie*, Morbid anatomy, etc. 1793. p. 95.—*Autenrieth*, Anleitung für gerichtliche Aerzte. Tübingen. 1806. S. 67.—*Allan Burns*, Observ. on digestion of the stomach after death. Medical and Surgical Journal. 1810.—*Jäger*, Ueber die Erweichung des Magengrunds. Hufeland's Journal. 1811. V. 3 ff. 1813. I. 15.—*Cruveilhier*, Anat. patholog. Livre X. 1821.—*Louis*, Du ramollissement avec amincissement, etc. Archiv. général. Tom. V. 5.—*Billard*, Die Schleimhaut des Magens und Darmkanals. übers. von Urban. S. 276.—*Elsässer*, Die Magenerweichung der Säuglinge. Stuttgart und Tübingen. 1846.—An excellent monograph, with a critical discussion of previous literature (*Nagel*, *Hauff*, *Rapp*, *Rokitansky*, *King*, *Carswell*, *Kreuser*, *Camerer*, *Engel*, *Heusinger*, *Broussais*, *Chaussier*, etc.).—*Virchow*, His Archiv. V. 359. 1853.—*Hirschsprung*, Den medfödte u. s. w. Diss. Kopenhagen. 1861. Referat in Schmidt's Jahrb. Bd. 117. 310.—*Rokitansky*, Aufl. III. der pathol. Anat. Band. 3. S. 178. 1861.—*F. W. Pavy*, Medic. Times and Gaz. Septbr. 1863.—*Thomas Langston* (Sudden death from softening of the stomach). Lancet. II. 17 Oct. 1863.—*Bamberger*, Krankheiten des chylopoëtischen Systems. 1864, with critical remarks and a bibliography. On the pathological anatomy, see: *Förster*, Spec. pathol. Anatomie. 1863.—*C. E. E. Hoffmann*, Ueber Erweichung und Durchbruch des Magens und der Speiseröhre. Virchow's Archiv. Bd. 44. Heft 3. 1868.—*W. Mayer*, Gastromalacia ante mortem. Diss. Inaugur. Erlang. Leipzig. 1871.

Pathological Anatomy.

At post-mortem examinations more or less softening is almost invariably found at the fundus of the stomach, and not unfrequently also at other portions of the organ.[1] Sometimes the mucous membrane merely appears to be of a pulpy consistence, and more easily separable from the muscularis than usual; sometimes the softening affects also the muscular and serous coats, or perhaps the wall of the stomach tears completely through on the attempt to take out the stomach or the spleen; at other times, on opening the abdomen, the organ is found to be already perforated, and its contents effused into the peritoneal cavity. Finally, in certain cases the softening extends from the stomach to the adjacent viscera—the spleen, liver, diaphragm, lungs and lower portion of the œsophagus. The parts thus secondarily involved in the softening are disintegrated, discolored, and *invariably free from any trace of reactive inflammation.*

In the *stomach* itself the softened spot presents various appearances, according to the greater or less amount of blood it contained when the softening process began. If the mucous membrane were at this time in a condition of anæmia, it becomes reduced to a "pale (colorless) gelatinous mass;" if, on the other hand, the vessels contained much blood the membrane is converted into a "blackish-brown pulpy mass, some of which, in the form of matter looking like coffee-grounds, becomes mixed with the contents of the stomach" (Rokitansky). Gastromalacia, accordingly, presents two leading forms: a *gelatinous* and a *black* variety.

The softened spot is characterized by the absence of a sharp border, and of all signs of inflammation at its periphery; while in advanced degrees of the malacial process the softened spot is replaced by a "hole which is bounded by a liquefied, smooth or fatty border."

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[1] Thus in Allan Burns' celebrated case (see below) the *anterior* wall of the stomach was the part chiefly affected.

The changes described are usually found in the bodies of children, especially in those who have had cholera infantum, and particularly at the period of weaning when the change in diet is very apt to excite fermentative processes in the stomach. Whether other diseases, such as brain affections, tuberculosis, pneumonia, etc., induce a predisposition to the occurrence of gastromalacia, cannot be determined until larger statistics are collected on this point. Softening of the stomach is found also in *adults*, not only after the occurrence of previous disease, but even in those who, while in the midst of perfect health, have met with a violent death shortly after taking food.

Causes of Softening of the Stomach.

Hunter was the first to express the opinion that the pulpy *softening of the wall of the stomach* found at autopsies is the *result of the dissolving action of the gastric juice* after death, and that the parts attacked are those at which the gastric juice accumulates. In the recumbent position of the cadaver most of the fluid gravitates to the cardia, and here, therefore, the lesion will generally be most marked; but as the softening process spreads, other portions of the stomach and even other viscera may become eroded.

This opinion of Hunter was confirmed by some observations of M. Baillie and Allan Burns, the latter of whom was the first to investigate this pathological question experimentally.

Two days after death he opened the body of a young emaciated girl who had died of scrofulous enlargement of the mesenteric glands. The empty stomach was found to be dissolved on its anterior aspect; the hole was longitudinal in shape, between one and two inches in size, with friable, spongy, soft edges. The liver was in contact with these parts, but, as he demonstrated to his pupils, had not been in the least affected. He now replaced all the parts in their usual position, sewed up the abdomen, and kept the body in a cool place. The abdomen was again opened two days afterwards. *The liver, where it lay in contact with the dissolved portion of the stomach, was now softened to a considerable depth,* and the adjacent peritoneum was entirely dissolved. All other portions of the liver presented their usual firmness. *Nowhere in the body was there any trace of decomposition.* The *posterior* wall of the stomach was also dissolved, as far as the abdominal wall, into a pulpy glutinous substance. Since the body had all the time lain in the recumbent position, and since, therefore, the gastric juice could not have come in contact with the softened spot lying anterior to the pylorus, A. Burns, after two other similar experiments, concluded that the solution had been effected, not by the gastric juice which had been poured out into the stomach, but by that which had been retained in the secreting vessels.

In the year 1811 Jaeger published a treatise "On Softening of the Fundus of the Stomach, etc.," in which he laid down the proposition that the softening found in the stomach after death is "unmistakably connected" with a definite morbid process running its course *intra vitam;* and the fact that in several cases of children his diagnosis during life was actually confirmed by the autopsy, *seemed to justify the hope that pathology had gained a new disease—"softening of the stomach."*

Jaeger's description of the symptoms is briefly as follows: Loss of appetite, hot hands, urgent thirst, very rapid pulse, restlessness, and expressions of pain make their appearance suddenly, or after several weeks of vomiting and diarrhœa; the face becomes pale and altered in appearance, and sometimes attacks of diarrhœa supervene. Then follow vomiting and convulsions, together with a dry, short cough, probably from irritation of the diaphragm, and finally death by collapse.

In his second edition, however, in the year 1813, Jaeger's vitalistic views had become much less pronounced; he now regarded the softening process as only commencing during life, and its extension, especially its spreading to the adjacent organs, as taking place after death. He finally sums up the results of his own observations and the descriptions of other writers as follows: "That the softening is preceded by an affection, which, operating through the nervous system, alters the secretion and the reaction of the intestinal canal towards its contents, and one result of which is an excessive formation of *acetic acid*, which is the immediate cause of the softening."

The French, as Louis, Cruveilhier, and other representative writers, likewise maintain the vital origin of gastromalacia. Thus, Cruveilhier draws a sharp distinction between the "ramollissement gélatiniforme," which occurs in a certain disease of nursing children, and the "ramollissement pultacé" or "cadavérique." Among the German champions of the vitalistic view Rokitansky has been the most weighty authority.

The most valuable article, however, upon this subject, is unquestionably the monograph of Elsässer (1846). This writer asserts positively, and without ambiguity, that *the softening of the stomach in nursing children is under all circumstances a post-mortem change.* The convincing nature of his experiments, and the force of his objections to the vitalistic theory have led to a general acceptance of his views in Germany.

His reasons for maintaining the *cadaverous* nature of this morbid process are as convincing to-day as ever, and may be briefly stated as follows:

The *location* of the softening depends upon the position of the cadaver, and may be changed at will to different parts of the stomach, according as the body is placed immediately after death upon the back, abdomen, etc., since it is always the most inferior portion of the stomach which is softened. The *superficial extent* of the dissolved parts is proportioned to the size of the surface over which the wall of the stomach and the ingesta have been in contact with each other. *For the production of softening of the stomach a certain quantity of free acid is requisite;* the self-digestion of the stomach cannot under ordinary circumstances be effected by the acid secreted by the wall of the stomach for the digestion of its ingesta, because this quantity bears a direct relation to the amount of food to be digested. It is necessary, therefore, either that acid should be introduced from without, or that an acid fermentation should be set up in the stomach, such as occurs from the use of cane or milk sugar, and particularly milk; and this fermentation may take place either during life from an abnormal, weakened power of digestion, or after death. Softening does not occur in an empty stomach. Moreover, a *temperature*, approaching that of the stomach during life, is a necessary factor in the solvent action of the acid fermenting ingesta. It is well known that such a temperature is maintained for a considerable time in the cadaver; according to Nasse's experiments it was still 75° F. in a room at 55° after the lapse of fifteen hours. *The solvent action is counteracted* by common salt, and especially by *bile*, which has passed into the stomach. *Intestinal softening* occurs under essentially the same conditions as the softening of the stomach—that is, from contact of the intestinal wall with substances which have undergone an acid fermentation, and which, besides the acid, contain also the gastric pepsin which has been hurried by a morbidly increased peristalsis into the lower parts of the intestine, and has failed to be neutralized by the alkaline secretion of the intestines. The intestinal softening does not occur, therefore, with a normal digestion, but only with a morbid condition of the alimentary canal, as distinguished from the softening of the stomach, which may be found even with a normal digestive function up to the time of death. *Softening of the stomach is more frequently found in the bodies of nursing children*, because in them the coats of the stomach are more delicate, and the food is chiefly composed of substances such as milk, sugar, etc., which, from the absence of salt, are susceptible of an acid fermentation, and which, moreover, are usually administered even up to the moment of death; and, finally, because cholera morbus, with its processes of acid fermentation, is more common at this age than at other periods of life. *An empty stomach is less soluble in acid menstrua than a stomach engaged in the process of digestion,* a fact which shows that the softening is favored by the physiological condition of the tissue of the organ. Moreover, if the gastromalacia occurred during life, it is surprising that hæmatemesis is never observed, and that in the cadaver no signs whatever of inflammation are to be detected in the neighborhood of the softened part.

These propositions of Elsässer have contributed materially to the settlement of the question in regard to the nature of gastromalacia, and most recent pathologists, Brinton, Förster, Oppolzer, Virchow, Bamberger, and others, have adopted the same view, that *softening of the stomach is to be interpreted as a cadaveric symptom.*

The facts above mentioned are so convincing that there can no longer be any question as to the general truth of this view. *Still, no evidence has been adduced to show that the inception of gastromalacia during the last period of life is absolutely impossible.* In fact, the case observed by C. E. E. Hoffmann, in which, three hours after death from a violent attack of coughing, ruptures of the stomach and œsophagus were found at the autopsy, speaks very strongly in favor of the possibility of a vital gastromalacia. *All doubt, however, upon this point would be dissipated by a case which should show* INDISPUTABLY *that the simple softening found post-mortem had been preceded during life by the occurrence of perforation and its resultant symptoms.*

Such a case has, in fact, come under my own observation, and was diagnosticated by me as one of "perforation of the stomach."[1] The great importance of the case will probably be a sufficient excuse for giving the history *in extenso.*

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[1] The case occurred in Ziemssen's Clinique, and has been published by W. Mayer (Gastromalacia ante-mortem. Deutsch. Archiv f. klin. Med. Bd. IX. S. 105 ff. 1871), of which article the above is an abridgment. The analysis of the case, as there given, is strictly in accordance with the facts, and is much more complete than my own.

A man, thirty-seven years of age, was attacked in 1866 with a chronic gastric ulcer, the cicatricial contraction of which induced marked stricture of the cardia. In 1867 he entered the hospital, much reduced in nutrition, and unable to eat solid food. Cure from the use of the stomach tube and Carlsbad salt. In 1868, renewed attacks of ulceration, with comparatively rapid recovery each time. In 1869, good health.

November 9th, after eating and drinking to excess, he was suddenly taken ill, with symptoms of perforation of the gastro-intestinal canal. Death six hours and a half after the perforation was diagnosticated.

Autopsy: Cicatricial stricture of cardia and pylorus. At the fundus of the stomach was a softened perforated spot, nine ctm. in length. The peritoneal cavity was filled with air, and the intestines were everywhere covered by the contents of the stomach. Subcutaneous emphysema.

The patient, Johann Hoffmann, a waiter, thirty-seven years of age, came for the first time to the Erlangen Clinique, for treatment, on March 9, 1867.

While in uniform good health (with the exception of a previous attack of abdominal inflammation) he had been attacked, the past autumn, with frequent vomiting, especially after taking indigestible food—not directly after every meal, but particularly at night. Occasionally he vomited blood, and passed coal-black stools. About three weeks ago the very severe pains disappeared almost entirely, and were replaced by a sensation as if the food failed to pass into the stomach. Immediately after eating, the food regurgitated, without any direct effort at vomiting. He had therefore confined himself almost entirely to fluid nourishment; but even this sometimes failed to be retained, so that he had become very weak and much emaciated. It was ascertained, moreover, that his father had died of an affection of the stomach, which had lasted about a year.

Present state, March 9, 1867:

A very emaciated individual, of medium size; pallid appearance; relaxed skin and musculature; no panniculus adiposus; slight œdema. Very great muscular weakness, so that even the slightest work is impossible.

Fluids are swallowed without difficulty, but food of a more solid consistence fails to pass down. Epigastrium not tender on pressure; no retraction, nor protuberance, and no greater resistance than usual. On examination with a medium-sized stomach-tube, the instrument encountered, at the level of the cardia, an obstruction, which could not be overcome. A sound, however, of five mm. in diameter, glided quite easily through the stricture, and afterwards an instrument of medium size succeeded in passing into the stomach.

In the right inguinal region was a small hernia, as large as a hen's egg, which had existed for six years, and which was retained by a truss.

Diagnosis.—Stricture of the cardia, resulting from cicatrization of a chronic round ulcer of the stomach. Anæmia from inanition.

By repeated catheterization of the cardia with instruments of increasing size, and by the use of Carlsbad salt, improvement was effected; but in the following year fresh ulcers were formed with the occurrence of hæmatemesis.

The patient was again put upon the same treatment, with Carlsbad salt and suitable nourishment, and the symptoms again gradually disappeared. After this time he remained in the hospital, where he was employed as an assistant nurse; his strength returned, and he tolerated without discomfort all kinds of food. From time to time, however, he was subject to attacks of gastric derangement, which consisted in severe pains in the epigastrium, with marked flatulent distention, entire loss of appetite, and general malaise, and which terminated either in the vomiting of a large quantity of very acid fluid or in diarrhœa.

On September 12, 1869, he was attacked by a pleuritis on the left side, from which he rapidly recovered. The effusion, which was very considerable, disappeared so far as to leave only a slight dulness over the posterior and inferior part of the chest, on the left side.

On November 9, 1869, after working as usual through the day, he was seized, at eleven o'clock at night, with an extremely severe pain, beginning in the hypogastrium, and extending, as he described it, over the whole abdomen.

It was ascertained from the statements of the patient, and from inquiries that were made, that he had been suffering for some three days past with disturbance of digestion, and that, the preceding noon, his abdomen had been somewhat distended by flatulence; moreover, that at about nine o'clock of the same evening he had eaten largely of brown bread, washed down with considerable quantities of very new, imperfectly fermented beer.

On examination, at eleven o'clock, the abdomen was much distended, rigidly tense, everywhere resonant; no dulness anywhere over the dependent parts; pulse very weak and rapid.

Sinapisms and injections of morphine had but little effect in relieving the intense pain, and the tympanites steadily increased. At the second examination, at three o'clock in the morning, it was found that *the dulness over the liver had entirely disappeared, and had been replaced by resonance.* The region of the hernial aperture was much distended, and the hernial sac in the scrotum was resonant on percussion.

As there was reason for apprehending strangulation of the hernia, which was evidently lying outside of the ring, I tried taxis, and succeeded within a few minutes, without resorting to force; *but, at the very moment of success, crepitation was noticed in the skin at the root of the penis, and a cutaneous emphysema arose here,* which, notwithstanding the application of the truss, spread within two hours over the whole abdomen and left side of the chest. The patient was pale and cyanotic; the sensorium but little disturbed; the respiration moderately accelerated, but not dyspnoic; great feebleness of the heart's action; pulse scarcely perceptible; extremities cold; face expressive of collapse; no cough; no vomiting; only nausea and attempts at vomiting.

The same symptoms continued, slight clonic contractions of the upper extremities took place, and at half-past six o'clock in the morning the patient died.

Autopsy, at half-past nine A.M., November 11, twenty-eight hours after death (Prof. Zenker):

Perforation of the stomach, with effusion of the contents of the stomach into the *abdominal cavity. Softening of the stomach. Cicatricial stricture of the cardia; cicatrices of the pylorus and duodenum. Partial maceration of the peritoneum (serosa of the small intestine and hernial sac). Cutaneous emphysema. Hyperæmia of the left kidney. Remains of pleuritis on the left side.*

Cadaver of medium size, powerfully built, and well nourished; skin tense, of a dusky color, with large, dark cadaveric spots, much inflated and crepitant on the abdomen, left side of the chest and neck, upper parts of both thighs, scrotum, and penis; muscles well developed, powerful, bright red, and very rigid. The subcutaneous tissue over the symphysis was of a dirty-gray color, and infiltrated with serum.

Abdomen: On opening the cavity gas escaped: the protruding intestines were covered at some places with a thin, at others with a thicker, layer of dirty brown colored masses. In the pelvis were several ounces of a dirty brown, very oily fluid.

The *small intestine* was considerably distended so as to overlie the stomach and occupy the entire anterior abdominal space.

The *stomach* was very much contracted, pushed far backwards, and was covered on its cardiac half with a dirty red pulpy substance. *The pyloric portion* was closely adherent to the right curvature of the colon. *At the fundus was a laceration about nine ctm. long with a dark colored pulpy border.* Over the whole cardiac half the *mucous membrane* was thickened in the form of blackish emphysematous elevations, some of which were confluent and others circumscribed; while over the pyloric half the mucous membrane appeared smooth and pale. *The cardia* was contracted to an extraordinary degree; its lumen, in its tense condition, measured five mm. in diameter, and was surrounded by a radiating cicatrix one and a half ctm. in length. At the lower part of the *œsophagus* was a funnel-shaped contraction; the mucous membrane was immovable, smooth, with a dense epithelial layer, and the wall thickened for a length of from 3.5 ctm. to 6 mm. The *pylorus* was moderately narrowed, and just admitted the little finger; immediately in front of the pyloric valve was a cicatrix two mm. in length, and occupying a third part of the circumference, from which radiated several striæ.

At the beginning of the *duodenum* was an ill-defined cicatrized spot on the mucous membrane with short striæ; in the rest of the duodenum the mucous membrane was much injected, otherwise normal. The *small intestine* was throughout much distended, and contained a little yellow chyme; the mucous membrane for the most part presented a deep red injection, otherwise it was normal, except that in certain places the serosa was very soft and macerated. The *large intestine* contained a small quantity of pulpy brown fæces, and the mucous membrane was somewhat injected in spots.

Spleen of normal size, extremely soft, and its surface discolored; its substance pale, with spots of a dark violet color. *Liver:* right lobe attached to the diaphragm by numerous thin fibrous bands, and somewhat diminished in size in its vertical diameter; its substance of a uniform reddish brown color, and engorged with blood. The *gall bladder* contained quite a large quantity of pale watery bile.

Kidneys: the left considerably enlarged, surface smooth, and of a uniform dark violet color; on section the cortex and pyramids were extremely congested, and of a very dark violet color. The *right* kidney was much smaller, softer, and less congested than the left.

On the right side was a large inguinal hernial sac with a wide opening, containing a *considerable quantity of a dirty gray greasy ichorous fluid;* the *hernial* sac itself was softened to a pulpy consistence, and had broken down into an ichorous matter.

Bladder contracted, containing a little pale gray watery fluid.

Chest: the diaphragm on the left side reached to the fourth rib. The *left lung* was everywhere quite firmly adherent, but at the posterior part of the inferior lobe there was an extremely firm adhesion by means of a thick cicatrix of cartilaginous hardness. The right lung was everywhere free.

The preceding case is of great importance as regards the theory of softening of the stomach, for the reason that the accidental laceration of the hernial sac in the effort at reduction, and the enormous cutaneous emphysema which followed, *demonstrated absolutely that the perforation of an air-containing abdominal viscus took place during life.* The autopsy showed an unmistakable softening of the stomach with a complete destruction of the wall, in the form of "a laceration nine ctm. in length at the fundus, with a blackish, pulpy, softened border." *Furthermore, the intestinal convolutions were found covered with the contents of the stomach* ("with thinner or thicker layers of dirty-brown colored masses)." If the perforation had not occurred until after death, the fluid from the stomach would not have been so *uniformly* distributed over the surface of all the intestinal convolutions. This result was due to the peristaltic movements of the intestines after the contents of the stomach had escaped into the peritoneal cavity. The softening of the intestinal serosa, and particularly of the hernial sac, was also caused by the diffusion of the ingesta over the bowels; though, as regards the hernial sac, it is highly probable that the lesion had begun during life, because otherwise it would be difficult to explain why the hernial sac was instantly torn by a taxis, which was far from forcible. Moreover, the "blackish, emphysematous tumefactions," which were found over the entire cardiac region, as well as the "blackish, pulpy" appearance of the seat of laceration at the fundus, make it very probable that hemorrhages had taken place during life into the wall of the stomach.

These facts, taken in connection with the accepted views on the physiology of digestion, and especially with the results of Pavy's experiments on the self-digestion of the stomach,[1] seem to warrant the following conclusions in regard to the genesis of softening of the stomach:

> Softening of the stomach never occurs except when pepsin and acids are present in the stomach in sufficient quantity.
>
> During life, and under normal circumstances, the softening is prevented by the abundant circulation of alkaline blood in the wall of the stomach.
>
> After death—when the circulation is arrested—this protection is withdrawn, and softening must necessarily occur if suitable quantities of pepsin and acids be present at the time of death in the contents of the stomach, and if the body have not cooled too rapidly. The more gradually the temperature of the cadaver falls below the normal temperature of the living body, the greater is the activity of the pepsin, which, as is well known, digests most rapidly at 95° F., and loses its power when the temperature has sunk to about 39°.

Since even a small amount of pepsin is still effective, provided it be accompanied by a corresponding quantity of acid, the presence of large quantities of the latter in the stomach is a very important factor in the genesis of gastromalacia. All articles of food, therefore, like milk, sugar, etc., which readily undergo acid fermentation, are particularly apt, when taken just before death, to induce a post-mortem digestion of the stomach.

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[1] The main results of these experiments were: that parts of the extremities of a living animal introduced through a gastric fistula of another animal are digested; further, that the surface of the stomach can be deprived of a portion of its epithelium without the injured portion being thereby subjected to self-digestion, but that the latter takes place whenever the current of blood circulating in the wall of the stomach is interrupted, or the acidity of the gastric juice becomes excessive. It follows almost inevitably that it is the alkalinity of the blood circulating in the wall of the stomach which protects the organ from self-digestion, by neutralizing the acid in the mucous membrane. This immunity of the mucosa is due solely to the extraordinary vascularity of the wall of the stomach, while parts of the living body which contain comparatively less blood—such, for instance, as the foot of a frog—contain too little alkaline blood to long withstand the action of the acid gastric juice.

The failure to find softening, when the circumstances under which death has occurred and the body has been kept, would lead us to expect the lesion, may be explained by *the action of various causes, which either prevent its occurrence, or at least check its more marked development.* Among these—not to mention the exposure of the body to a low temperature—are *the presence of bile*, metallic salts, etc., and *the accumulation of large quantities of peptones* in the interior of the stomach. The ingress of bile into the stomach, which is very apt to occur in cholera infantum, from the repeated acts of vomiting, produces a precipitation of the pepsin, and thus checks the digestive action of the gastric juice on the wall of the stomach. This result is favored also by the fact that, after death, the absorption of the *peptones* no longer continues with the same facility as during life.

Softening of the stomach may also begin before death, in fact, may even proceed to complete perforation; still, such instances are extremely rare in comparison with the cases in which it is a post-mortem lesion. Its occurrence during life is undoubtedly possible only through the co-operation of several predisposing causes, such as the excessive formation of acids from imperfect digestion, their retention in the stomach, by narrowing of the orifices, or other causes, thinning of the wall of the stomach by gaseous distention, and delicacy or other morbid conditions of the texture of the stomach. Daily experience shows, however, that these favoring conditions are all insufficient of themselves to produce a softening of the stomach, and that this result does not occur until the incessantly renewed, counteracting influence of the alkaline blood in the wall of the stomach is withdrawn by an arrest of the circulation, such as results from hemorrhages, arterial embolism, etc. Naturally, also, the self-digestion will be more intense and more extensive, the more the causes above mentioned combine with this factor to produce the result in question. For otherwise it would be difficult to explain why in the rare cases of ante-mortem gastromalacia the self-digestion, when once begun, should not be limited, as it is in the formation of ulcers, but should become diffuse and perhaps induce rapid perforation. Possibly, moreover, a *complete arrest* of the circulation is unnecessary; it may be that a diminished supply of alkaline blood in the wall of the stomach, such as may be supposed to occur in collapse or during the last hours of life, is sufficient, in connection with the auxiliary cause above mentioned, to initiate a process which, under favorable conditions, may complete its full development after death.

The fact that, in cases of apparently vital gastromalacia, just as in those of post-mortem origin, it is the most dependent parts of the stomach which are softened, is no certain indication of the cadaveric character of the process, because it is extremely probable that shortly before death the contractions of the stomach become more feeble. The vomiting will consequently cease, the acid chyme accumulate at the fundus of the stomach, and softening take place at that point.

Another disputed point which deserves special mention is the reciprocal relation between diseases of the brain and softening of the stomach. In the early part of the present century Jaeger expressed the opinion that gastromalacia owes its origin ultimately to a morbid condition of the nervous system, and after him others frequently noticed the occurrence of softening of the stomach coincidently with affections of the brain (basilar meningitis, encephalitis, etc.).

A compilation of cases of gastromalacia made by me from the records of the Pathologico-anatomical Institute of this city showed that, out of 2,053 autopsies, there were 53 in which marked softening of the stomach was expressly mentioned. Of these 53 no less than 18, or a full third, were associated with disease of the brain. This number of cases is, of course, too small to warrant a positive conclusion, but the proportion is certainly very striking.

The question, whether this frequent association of the two lesions in the dead body is an accidental or a causal one cannot be decided with our present material. Still, I would call attention to the fact that Rokitansky, with his abundant experience, entertains no doubt of the intimate relation between the two processes, nor of the ante-mortem origin of the softenings of the stomach found after death in connection with brain diseases. Moreover, the same inference seems to be warranted by the experiments of Schiff, in which an injury to certain parts of the brain resulted in hemorrhagic infiltrations and local softening of the wall of the stomach.

Hemorrhage of the Stomach.

The most complete account of the older literature will be found in *Plouquet's* Literat. med. digest. 1808, under the articles Haematemesis und Melaena. —*Kuhk*, Jahresbericht der Berliner Charité vom Jahre 1832. Rust's Magaz. 1835. S. 302.—*Dassen*, Ueber Morbus niger. Schmidt's J.-B. 22. S. 172. 1838.— *Willigk*, Sectionsergebnisse, etc. Prager Vierteljahrschrift. 1853. Bd. II. S. 1; 1865. Bd. IV. S. 18; 1856. Bd. II. S. 1. ff.—*Osborne*, On some leading facts, etc. Dublin Journal. Febr. Vol. XV. p. 107. 1853.—*Rowland*, Tödtliches Blutbrechen, etc. Union. 19. 1857. Schmidt's J.-B. 96. S. 61.—*Le Diberder u. Fauvel*, Oesophagusvaricen mit Blutung. Gaz. hebd. V. 13. 1858.—*Bristowe*, Varicose ulcer of the œsophagus, etc. Transact. of the Path. Soc. Lond. Vol. VIII. p. 175. —*Liljebörn*, Hygiea. Band 19. Schmidt's J. -B. 102. S.296.—*A. v. Graefe*, 2 Fälle von plötzlicher und incurabler Amaurose. Archiv für Ophthalmol. VII. 2. S. 143. 1860. — *Fikentscher*, Fall von plötzl. Amaurose, etc. Archiv für Ophthalm. VIII. 209.—*Handfield Jones*, On hæmatemesis, etc. Med. Chir. Transact. XLIII. p. 353.—*Baeumler*, Ein Fall von Verschliessung der V. cav. inf. u. s. w. Deutsche Klinik. 1862. No. 12 u. 14.—*Binz*, Perforirendes Magengeschwür bei Neugeborenen. Berliner klin. Wochenschr. 1865. Nos. 15, 16.—*Emil Rollet*, Wiener. medic. Zeitschrift. No. 99-101. Canstatt's Jahresber. 1866. I. S. 207.—*Murchison*, A treatise on the continued fevers of Great Britain. 1862.— *Walter Jones*, Fatal hemorrhage in cirrhosis of the liver, Lancet. Febr. 1868.—*Hutchinson*, Amaurosis, etc. Ophthalm. Hosp. Rep. VI. 3. 218.—*Spiegelberg*, Zur Melaena neonatorum. Jahrb. der Kinderheilk. II. 1869.—*Boon*, Annales de la Societ. de Med. d'Anvers. Decbr. Ref. Canstatt's J.-B. II. 218. 1870.—*Michel*, Transfusion mit Erfolg nach einer profusen Magenblutung, etc. Berliner klin. Wochenschrift. 1870. No. 47.— *Wilson Fox*, Diseases of the stomach, 1872, with an account of the literature of the subject, p. 204.—*Landau*, Ueber Melaena. Habilitationsschrift. Breslau. 1874.

Historical Introduction.

The Hippocratic school of medicine, as is well known, taught that the human body contains blood, mucus, yellow and black bile ("χολην ξανϑην και μελαιναν," Hippokratis Lib. de nat. hom. autore Foesio. p. 225). Black-looking masses of blood in the vomited matters or in the stools, the dark color of which was due to their long retention in the stomach, were therefore regarded as black bile, and the disease of which this was the symptom was called *melaena* or *morbus niger*, a full description of which is given in the second book of Hippocrates' "De Morbis," p. 486. The vomiting of black blood was called "*vomitus melancholicus*," and was referred to the spleen, because this organ was supposed to secrete a "succus melancholicus coloris nigricantis." And so when the autopsy[1] in cases of death from melæna revealed an alteration in the form and color (black) of the spleen, the dependence of the black vomiting upon morbid conditions of the spleen seemed placed beyond doubt. But gradually the conviction began to gain ground that this dark matter discharged by vomiting or stool was neither a secretion from the spleen nor bile from the liver, but only altered blood. This opinion, which is the one now accepted, was based upon the observation that the quantity of black vomited matters was too large to be regarded as a secretion of the liver or spleen, and furthermore, upon the discovery in certain post-mortem examinations that while the liver and spleen revealed no lesion, the same black matter, which the patient had vomited during life, could be seen, on the application of "gentle" pressure to the wall of the stomach, to directly "exude" from the gastric vessels. Finally, Portal[2] subjected this "bilis atra" to a physico-physiological examination, and was led by various considerations—above all, by the absence of any bitter taste—to the following conclusion: "The *black matter* discharged by vomiting and stool, sometimes only by vomiting, less frequently only by stool, is *not of the nature of bile, but is blood*, which acquires its black color in the alimentary canal, unquestionably because, being no longer in contact with oxygen gas, it becomes carbonated, etc."

Thus by the beginning of the present century the "black bile" had disappeared from pathology, and the morbus niger had been referred to the category of gastric and intestinal hemorrhages.

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[1] See *e.g.*, *Simson*, De re medic. diss. Edinb. 1726. p. 154; *Hertius* (F. Hoffmann), De morbo nigro Hippokr. diss. Halæ. 1701. § 8.

[2] Sammlung auserlesener Abhandl. 19. Band. S. 323. ff. 1800.

Pathological Anatomy.

In persons dying from hæmatemesis there is usually found, in addition to general anæmia, a large quantity of fluid or coagulated blood in the stomach. According to the degree to which the effused blood has been acted upon by the gastric juice, the color varies from a *brown* to a black, owing to the conversion of the hæmoglobin by the gastric acids into hæmatin, which, as is well known, has a brown appearance in an acid solution.

The *quantity* of blood found in the stomach varies considerably, from a few particles of a coffee-ground appearance to large coagula, which fill up the entire cavity, and restore the contour of the organ.

In many cases the most careful examination fails to reveal the *source* of the hemorrhage. When the bleeding has been purely capillary, the preceding congestion of the mucous membrane may have been so relieved by the hemorrhage that the surface appears as pale as other mucous membranes. In other cases the tissue of the mucous membrane is found infiltrated with blood, or covered with hemorrhagic erosions; under these circumstances the quantity of effused blood found in the cavity itself is usually quite small. The first of these lesions—the *hemorrhagic infiltration*—consists of bluish red spots of larger or smaller size, and is very often accompanied by the "hemorrhagic erosions." The latter are composed of small round or oblong excavations in the mucous membrane, which itself presents an unaltered appearance, except that it is relaxed, pulpy, red, or perhaps pale. The erosions are sometimes very few in number; at other times the mucous membrane is covered with them, especially in the pyloric half of the stomach. Their favorite situation is on the top of the folds of mucous membrane, perhaps because the pressure from their juxtaposition impedes the reflux of blood. Usually the solution of continuity in the mucous membrane is not detected until the adherent blackish-brown coagulum has been removed.

According to Willigk's reports, hemorrhagic erosions are found in not quite two per cent. of post-mortem examinations (81 times out of 4,547, or about 1.8 per cent.). Both sexes are affected in about equal proportions (in 2,114 autopsies of males, erosions were found 36 times=1.7 per cent.; in 2,433 autopsies of females, 45 times=1.8 per cent.).

In another series of cases the hemorrhage is found to proceed from the *erosion of a large vessel*, and the coagulum adhering to the mucous membrane can be traced into the ruptured vessel. Sometimes this vessel lies upon the site of a recent lesion of the mucous membrane, a gastric ulcer, or a carcinoma ; at other times a copious hemorrhage proceeds from the rupture of a varicose vein, or of an aneurism of a vessel in the neighborhood. Finally, the blood found in the stomach after death occasionally comes, not from the wall of the stomach itself, but from hemorrhage in the cavities of the nose and mouth, in the œsophagus or in the air-passages, the blood passing into the stomach by swallowing.

Etiology and Pathogenesis.

The immediate cause of the hemorrhage is always a *loss of resisting power on the part of the wall of the vessel to the blood-pressure within the vessel.* The conditions under which this disproportion occurs are of the most diverse character. Usually the two factors—*the increase of the blood-pressure and the alteration in the wall of the vessel*—act in combination, and, therefore, in our classification of the causes of gastric hemorrhage under the one or the other of these categories, it is to be understood, without our constantly referring to it, that the other etiological factor may also be present.

Until recently a solution of continuity in the wall of the vessel was regarded as indispensable to the occurrence of hemorrhage; but the recent discoveries of Cohnheim in regard to venous stasis and inflammation have shown that this lesion is no longer a prerequisite, at least not for the circumscribed parenchymatous hemorrhages with which we have become familiar in the case of hemorrhagic erosions, and whose origin in a simple diapedesis of red blood corpuscles must now be accepted as demonstrated in the majority of instances. The special causes of gastric hemorrhage may be classified under the following groups.

1. *Direct injury* to the *gastric vessels* by means of wounds to the wall of the stomach (for example, by sharp bodies which have been swallowed), or by means of corrosive chemical substances. Under the latter head may be classed even the normal gastric juice, when derangements of the circulation have given it an opportunity to exert its corrosive power. This is the explanation of the gastric hemorrhages, which occur in simple ulcer of the stomach and in carcinoma, and which, in the latter affection, are favored to an important degree by the formation of thrombi, by local stases, and by the delicacy of the newly formed vessels.

2. *Disease of the wall of the vessel*, *e. g.*, *varices*, *aneurisms* of large arteries in the neighborhood, which become adherent to the wall of the stomach and perforate it, and degenerative processes in the gastric vessels themselves, such as fatty degeneration or *atheroma.* Probably, also, the hemorrhages which occur in *severe inflammation of the stomach* should be regarded as dependent upon an alteration in the walls of the vessels.

3. *Venous stases in the wall of the stomach*, leading to a diapedesis of the red blood corpuscles, or to ruptures of the capillaries with extravasation of blood upon the free surface of the organ. The immediate cause of these stases is an obstruction to the portal circulation, either in the liver or on this side of it, such as occurs in *pylephlebitis*, in pressure on the portal vein by tumors near the transverse fissure, in *cirrhosis of the liver* with constriction of the portal vessels by neoplastic connective tissue, in *icterus* when the branches of the portal vein are compressed in consequence of biliary obstruction, in the *pigment liver* when the portal vessels in the liver are occluded by flakes of pigment, in *multilocular hydatids of the liver ;* finally, we may here include also the hæmatemesis which occurs when the cessation of a regularly recurring hemorrhoidal bleeding has led to stasis and increased lateral pressure in the inferior mesenteric vein, and thus indirectly in the veins of the stomach. In stenosis of the vena cava inferior beyond the entrance of the hepatic veins, and also in heart and lung diseases, the cause of the gastric hemorrhages is more remote, because here the stasis occurs primarily in the hepatic veins, and from them extends secondarily into the radicles of the portal vein. A portion of the venous blood of the

stomach, however—viz., the portion in the area of distribution of the superior gastric vein—does not flow through this vessel into the portal vein, but into the system of the cava through the inferior œsophageal and diaphragmatic veins, which communicate with branches of the superior gastric, so that stases which originate at the heart may extend directly to the wall of the stomach without taking the circuitous route of the hepatic circulation.

In this class of hæmatemeses depending upon venous congestion are to be included also some of the gastric hemorrhages observed in *new-born children* (*melæna neonatorum*) during the first few days of life, or at least during the course of the first month. If in the period immediately following birth the respiration be imperfectly established, the right heart will have difficulty in emptying itself through the pulmonary artery, and thus stasis in the gastric vessels and hemorrhage may readily be produced in the way above described.

The majority of cases of melæna neonatorum are, however, probably due to the existence of gastric or duodenal ulcers. At least, this source of hæmatemesis in new-born children has of late been repeatedly demonstrated at autopsies. Thus Buhl and Hecker, Spiegelberg, Binz, and Landau found in the examinations made by them sometimes duodenal, at other times gastric ulcers covered with coagula of blood. In Landau's preparation there was also a thrombosis of the umbilical vein. When the expansion of the lung takes place imperfectly in the new-born child, small coagula may readily escape from the umbilical vein through the ductus arteriosus Botalli into the descending aorta and gastric arteries, and may thus, according to Landau, lead to the formation of gastric ulcers by an embolic method similar to that in which the round ulcer is produced in adults. Embolism of a gastric artery may occur in another way: a thrombus, forming originally in the umbilical vein, is driven through the ductus venosus Arantii into the ductus Botalli, becomes impacted there, and gives rise to further emboli. *According to Landau, therefore, the ulcers in the stomach and duodenum, which are the immediate cause of melæna, are produced post partum through emboli coming either from a primary thrombus of the umbilical vein, or from a secondary thrombus of the ductus Botalli*—the thrombosis itself being occasioned by a feeble action of the heart, by a long delay in the establishment of respiration, or by compression of the umbilical cord.

Whether an *arterial congestion* ever exerts sufficient pressure to rupture the walls of the capillaries, I very much question; and yet it is not to be denied that a sudden suppression of the menses may sometimes lead to congestions as well as to hemorrhages in various organs, particularly the stomach.

It is well known, also, that in cases of chronic amenorrhœa, regularly recurring hemorrhages, accompanied by molimina mensium, may occur in the stomach as well as in other organs. Still, the discoveries of Cohnheim in regard to congestion, stasis, etc., have shown the necessity of caution in our interpretation of these hemorrhages from "collateral fluxion." It becomes necessary to inquire, therefore, whether such congestive hemorrhages may not be due to a morbid deficiency of resisting power on the part of the vessels, and, above all, whether in cases of vicarious menstruation an important part in the causation of the hemorrhage may not be played by the *chlorosis*, which itself induces a fatty metamorphosis of the walls of the vessels, and which, as Virchow[1] has recently specially insisted, produces increased vascular pressure by the hypoplasia of the vessels which attends this affection—both of which conditions evidently favor a rupture of the gastric vessels.

The gastric hemorrhages, which occur in cases of *hemorrhagic diathesis*, are probably also to be explained by a hypoplasia of the circulatory apparatus in connection with a relative excess of blood ; at least, the results of Virchow's autopsies are very favorable to such an hypothesis.

4. The causes of the gastric hemorrhages which occur in *scorbutus*, *yellow fever*, and *acute yellow atrophy of the liver*, are at present unknown. Experiments certainly seem to show that the extravasations in these cases may be directly due to qualitative alterations of the blood itself, without any secondary mal-nutrition of the wall of the vessel. In scorbutus, however, notwithstanding numerous analyses, no positive evidence of such an alteration in the blood has ever yet been obtained. In yellow fever and malignant jaundice, the hæmatemesis as well as the acute hemorrhagic diathesis may be favored by the dissolution of the red blood corpuscles by the bile-acids ; but this by no means wholly explains the pathogenesis of hæmatemesis in these severe hepatic diseases, because, especially in acute yellow atrophy of the liver, the enlargement of the spleen in connection with the portal hemorrhages points unmistakably to the cooperation of mechanical conditions, particularly obstructions in the portal circulation.

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[1] Ueber die Chlorose, etc. Berlin. 1872.

To an altered composition of the blood may be referred also the gastric hemorrhages which occur in *chronic diseases of the spleen.* It is probable that the diminution in the number of red blood corpuscles in these affections, by inducing a general malnutrition of the body, occasions also a loss of resisting power on the part of walls of the vessels, and thus favors the occurrence of rupture. In the same way we may perhaps explain the gastric hemorrhages which are observed during the course of exhausting diseases, typhus, recurrent fevers, etc.

In some cases the hemorrhage arises, not from the gastric vessels, but from some source above or below the stomach, and the blood after passing into the stomach, accumulates there until it finally excites vomiting. Thus hemorrhages from the nasal or pharyngeal cavities, or the swallowing of blood during birth in the case of new-born children, may give rise to an erroneous diagnosis of gastric hemorrhage or melæna neonatorum. So also in cases of bleeding high up in the intestines, the blood may regurgitate into the stomach, and induce hæmatemesis, as not very unfrequently occurs in duodenal ulceration, or in typhoid fever when the disease extends into the upper parts of the intestine.

The blood, whatever its source, if it remain for some time in the stomach, is altered by the gastric juice in the manner described above, and is at last either vomited or passes off in the stools as a black, tarry matter.

When the hemorrhages take place into the tissues of the wall of the stomach instead of upon the free surface, the so-called "hemorrhagic erosions" are ultimately produced, the genesis of which is still involved in some obscurity. Rokitansky's theory is that the hemorrhages and erosions are both occasioned by the solvent action of an excessively acid gastric juice, or, as he himself explains it: "A change, beginning in hyperæmia, takes place in the secretion of the pepsin glands, whereby the secretion becomes abnormally acid, and dissolves first the secretory organs, and then the tissue and vessels, thus inducing hemorrhage." This explanation is, however, unsatisfactory, because it presupposes an undemonstrated *functional* derangement of the glands as the essential cause of the pathologico-anatomical lesion. If we recall the fact that every considerable infiltration of the gastric parenchyma with blood is dependent upon an obstruction of the vessels, and that every interruption of the circulation occasions a diminution in the alkalinity of the affected part,[1] it is apparent that a loss of substance—an "erosion"—will be the natural result of the action of the gastric juice upon the part which has been infiltrated with blood. Now, the wall of the stomach is specially liable to the occurrence of a hemorrhagic infiltration. The very arrangement of the capillaries, their interposition between single venous radicles on one side, and double arterial radicles on the other, impedes to a certain extent the flow of the capillary blood. The contraction of the muscles, especially during digestion, has a similar effect. If to these causes we add also a continuous passive congestion from remote causes, such as diseases of the liver (cirrhosis), cardiac diseases, respiratory diseases with vascular disturbances (emphysema, pneumonia, etc.), or transient obstruction of the circulation, as in the *act of vomiting*, it is easy to see how a laceration of the wall of a vessel and the formation of a hemorrhagic infiltration can be produced. The same explanation will apply also to the very frequent occurrence of erosions in gastritis. As soon as the vessels have become seriously altered by this disease, a profuse diapedesis of red blood corpuscles takes place from the capillary membranes, under the co-operating influence of the above-mentioned causes, and stagnation is produced. But when a considerable quantity of blood is extravasated into the coats of the stomach, the normal circulation in the district of the hemorrhagic infiltration is interrupted, and the *continuously* renewed neutralization of the gastric juice by the alkaline blood is thereby prevented. In this limited portion of the wall of the stomach there now necessarily ensues a "self-digestion," which manifests itself in the form of a hemorrhagic erosion on the inner surface of the stomach.

If Willigk's compilation of cases, in which hemorrhagic erosions were found after death (81 out of 4,547 autopsies) be analyzed with reference to the relative numbers at different ages, it will be found that, although the total number of cases in the two sexes is about the same, women exhibit an extraordinary disposition to the disease between forty and fifty years of age. Thus, while between ten and eighty years the frequency of the affection for each decade averages, without great variations, about one to sixty—that is, hemorrhagic erosions are found once in every sixty autopsies—the proportion suddenly rises in the fifth decade to one in twenty-seven. This result I believe to be undoubtedly due to the cessation of the menses at this period, and to the resulting congestions in various parts of the body.

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[1] For the details on this point see the chapter on the Etiology of Gastric Ulcer.

The absolute *frequency* of gastric hemorrhage can be estimated only approximately, and is a question of subordinate importance. Handfield Jones has observed hæmatemesis seventy-two times in ten thousand cases of disease. Of more practical importance is a tabulation based upon the relative frequency of the causes of gastric hemorrhage. Such a scale is given by Bamberger, who has arranged the diseases which occasion this symptom in the following descending order: simple ulcer, carcinoma, hemorrhagic erosion and mechanical obstruction of the vessels, poisoning, wounds, and foreign bodies, dyscrasiæ (scorbutus), inflammations of the mucous membrane, rupture of large aneurisms (aortic, cœliac), and vicarious hemorrhages.

The *female sex* appears to suffer from hæmatemesis more frequently than the male. According to Handfield Jones's compilation, the proportion is seventy-four per cent. females to twenty-six per cent. males—a proportion which our previous consideration of the causes of the disease would have led us to expect.

Hæmatemesis occurs at every *age*, but certain forms are met with only at certain periods of life, as, for instance, the melæna neonatorum, and the hemorrhages of the gastric mucous membrane which are connected with menstruation, chlorosis, and the menopause.

The hemorrhage usually takes place spontaneously, but in some cases its occurrence seems to be due to certain *exciting causes*, which by themselves are insufficient to induce hemorrhage in a sound gastric mucous membrane, but are able to effect a rupture when a tendency to hemorrhage already exists on the part of the vessels. Under this head may be enumerated mental excitement, strains and concussions of the body, overdistention of the stomach, etc.

Symptomatology.

In some cases gastric hemorrhage is unattended by any symptoms. Thus, if the blood extravasated into the cavity of the stomach be small in amount and be discharged, not by vomiting but in the stools, which are naturally not often inspected by the patient, the occurrence of a gastric hemorrhage may be entirely unsuspected; and it is quite probable that in severe gastric catarrhs, in carcinoma of the stomach, and in other diseases attended by slight gastric hemorrhages, such extravasations occur much more frequently than is commonly supposed. The same is true, also, for the great majority of hemorrhagic erosions, which are ordinarily first discovered accidentally at the autopsy.

When recognizable quantities of blood are discharged by vomiting or stool, the nature of the symptoms will depend upon the severity of the hemorrhage. If the loss of blood be moderate, the previous symptoms of the disease or the apparently healthy condition of the patient will not be materially changed; if, however, large quantities of blood are poured out into the stomach, symptoms arise which indicate unmistakably the serious nature of the accident. These symptoms are usually as follows:

After having for some time previously suffered from the usual symptoms of gastric ulcer, cirrhosis of the liver, etc. (see above), the patient is now attacked with a sensation of pressure, fulness, warmth, pulsation, and similar feelings in the epigastrium, accompanied by nausea, an unpleasant insipid taste, and the sensation as if a fluid were rising into the œsophagus. As the blood is poured out into the cavity of the stomach, direct symptoms of hemorrhage occur—giddiness, ringing in the ears, glimmering before the eyes, and faintness, while the pulse becomes frequent and small, or may entirely disappear. The nausea now increases, a violent attack of vomiting ensues, and the blood is discharged in a single sudden gush or intermittingly, through the mouth or through both the mouth and the nose. At the same time there is almost always some coughing from irritation of the larynx by blood which has entered the cavity during the act of vomiting. This fact should be borne in mind, because when the patient is afterwards questioned as to whether the blood was vomited or coughed up, he is very apt to suppose, from the coughing which attended the hæmatemesis, that the blood was really expectorated. In very rare cases ("internal hemorrhage") the blood is neither vomited nor coughed up, but remains in the stomach, over which the percussion note becomes more and more dull, until, finally, the blood passes off in the stools; the symptoms in the meantime being those of a profuse concealed hemorrhage, the patient becoming pale and cold, collapsed, pulseless, and affected with tremors or convulsions. If the hemorrhage be very profuse, immediate death may ensue under these symptoms, whether the blood remains in the stomach or be discharged externally.

The *vomited blood* presents different appearances, according to the duration of its detention in the stomach. If it be vomited immediately, it has the usual appearance of arterial or venous blood. Usually it is partly fluid, partly in coagulated masses—in the latter form, of course, only when the blood has remained in the stomach for some time before it was vomited. Generally its appearance is similar to that of coffee-grounds or chocolate, or perhaps it may still better be compared to that of moistened soot. This *change in color* is due to the action of the gastric juice upon the red hæmoglobin of the blood, which, under the influence of acids, becomes decomposed into globulin and *hæmatin*, the latter giving to the vomited matters its brown-red color. This alteration in color is consequently always an indication that the blood has for some time been in contact with the gastric juice, and although, in my opinion, it cannot be inferred merely from this appearance that the hemorrhage is arrested, still it is at least safe to conclude that no very *profuse* fresh bleeding is taking place. The *quantity* of the vomited blood varies considerably; sometimes it consists only of a few hemorrhagic specks, while at other times several pounds are vomited. If the vomiting be severe and the stomach be filled with food at the time of the hemorrhage, portions of the food, bile, mucus, etc., will be found in addition to the blood. Sooner or later after the attack, generally on the following days, colicky pains almost invariably occur, and blood makes its appearance also in the stools in the form of a black tarry substance.

In severe gastric hemorrhages the *physical* examination shows the abdomen to be distended by the blood which has accumulated in the stomach; but this sign disappears as soon as the blood is discharged by vomiting, and the dulness on percussion in the epigastrium is again replaced by a tympanitic sound.

After the cessation of the hemorrhage, if the loss of blood have been considerable, the patient presents the usual symptoms of anæmia—paleness, coolness of the peripheral parts of the body, blackness before the eyes, giddiness, fainting, hallucinations, and dropsical swellings. Another very singular symptom, for which as yet no satisfactory explanation has been given, is the sudden occurrence of an incurable double *amaurosis*. The ophthalmoscopic examination in such cases reveals only a white coloration of the papillæ and thinning of the retinal arteries. The hypothesis, that the general anæmia, produced by the loss of blood, gives rise to an anæmic condition of the central portion of the optic nerves, resulting in a permanent alteration in these nerve centres, is lacking in plausibility because blindness may occur after hæmatemesis in the absence of marked anæmia, and, on the other, because the total blindness still remains after the symptoms of loss of blood have disappeared. Equally unsatisfactory is the hypothesis of an extravasation into the central portions of the optic apparatus, particularly the chiasma, taking place during the act of vomiting, because the amaurosis has been observed to occur independently of the act of emesis. A striking illustration is supplied by Fikentscher's case, in which the amaurosis occurred several days after a "concealed" gastric hemorrhage, which was unattended by vomiting, but was followed by tarry stools and attacks of fainting. Moreover, from the fact that the amaurosis, which follows large bleedings from other organs, disappears as soon as the patient regains his strength, it would seem that between certain parts of the brain and the vessels or nerves of the stomach there must exist a nexus, which we cannot at present explain, but for the existence of which we have at least some evidence in the experiments of Schiff, who found that gastric hemorrhages were produced by division of the optic thalami, etc.

Alarming as the symptoms of anæmia following a gastric hemorrhage often are, yet, with the exception of the unfortunate sequela just mentioned, they are of a transitory character. Indeed, when the bleeding has been occasioned by a morbid condition of the liver and vena portæ, the fundamental disease has often been noticed to be relieved by the occurrence of hæmatemesis, as shown, for instance, by a diminution of the ascites, and in cases of gastric ulcer by a disappearance of the attacks of cardialgia. On the other hand, when the patient has already been prostrated by previous diseases, etc., the loss of blood is an alarming symptom, not only as regards the immediate result, but also because it may induce long-continued dyspepsia, and protract the convalescence.

Since most of the morbid conditions which are attended by hæmatemesis are of a chronic character, *recurrences* of the hemorrhage are, as might be expected, quite common. There is an interesting form of recurrent hæmatemesis which occurs under the influence of malaria. Boon has very recently observed a case of this kind, in which the hemorrhages recurring on alternate days were permanently arrested by quinine.

As already mentioned, death may result in the attack from various causes, such as the erosion of a large vessel with uncontrollable hemorrhage, profuse frequently-recurring capillary bleedings—as in cirrhosis of the liver—suffocation in consequence of the escape of a large quantity of blood into the air-passages, or an inability on the part of the exhausted patient to get rid of the coagula in the throat which block up the entrance of the larynx. Death may also result from the long-continued anæmia following the hemorrhage.

Diagnosis.

Although a correct diagnosis can readily be made when the hæmatemesis is profuse, especially if one has the opportunity of witnessing the attack itself, yet in some cases it may be difficult to ascertain whether the blood was coughed up or vomited, or whether it came from the stomach or from the adjacent parts. Certainty is of course possible only when one can see the attack itself, or at least the vomited matters, and is not obliged to trust wholly to the usually under these circumstances exaggerated statements of the patient and his relatives.

The *first question* to be decided at the bedside in a case of supposed gastric hemorrhage is *whether the red or reddish-black substance vomited is really blood or not.* Usually a mere inspection will place the matter beyond doubt, but sometimes, especially if there be reason to suspect the patient of deception, a more careful investigation is necessary.

As a rule, a simple microscopical examination is sufficient to remove all doubt of the presence of blood in the vomited matters by the discovery of blood corpuscles. Should the latter be entirely absent, or fail to be detected, in consequence of their conversion into rudimentary forms from long retention in the stomach, the behavior of the red colored substance in the *spectroscope* will give us the desired information. Hoppe's plan is to warm the fluid with some dilute nitric acid for the sake of removing the coloring matters which interfere with the examination, filter, dissolve the precipitate in a very dilute solution of caustic soda, and then test in the spectrum. If hæmatin be present, an absorption band makes its appearance between C and D, nearer to C than to D. An absolute confirmation of the diagnosis may also be obtained by the *hæmin test* with chloride of sodium and concentrated acetic acid (for details, see Kühne, Physiol. Chem. S. 205).

After the actual presence of blood has been ascertained by one of these methods, the *second*, and much more difficult, question remains to be decided *whether the blood proceeds from the stomach or from some other source.* Occasionally the hæmatemesis is preceded by hemorrhage from the nose or cavity of the throat, and more or less blood is swallowed, which is afterwards vomited. Careful inspection of the fauces and nasal cavities should therefore never be neglected; and when there is reason to suspect that the blood proceeds from ulcerations in the œsophagus, this part should be examined with the œsophageal sound. When the hemorrhage comes from varices in the œsophagus, a differential diagnosis from gastric hemorrhage is impossible ; but fortunately such œsophageal hemorrhages are extraordinarily rare (le Diberder, Fauvel). In the majority of cases, on the contrary, the alternative lies between a *pulmonary* and a *gastric hemorrhage.* Every physician of much experience will admit that this is often a nice question to decide—in fact, one that frequently cannot be decided, especially when we have only the history of the case to guide us, and particularly, also, when we have to determine whether a hemorrhage, which occurred several years before the present attack, was an hæmoptysis or an hæmatemesis. Still greater difficulty may arise when the two acts occur together in the same case—that is, when the blood during an hæmoptysis is swallowed and afterwards vomited, or, on the other hand, when some of the blood during an hæmatemesis passes into the larynx and excites coughing with bloody expectoration. In such cases the first point to be ascertained is, whether the attack *begins* with an act of vomiting, or with paroxysms of cough—a point, however, in regard to which the patient, taken unawares as he is by the hemorrhage, can of course seldom give any accurate information after the attack is over. Fortunately, however, the chief distinctions between the two varieties of hemorrhage are sufficiently characteristic to enable us to decide upon the source of the blood, as will appear from the following points of contrast.

<table>
<tr><th>Hæmoptysis.</th><th>Hæmatemesis.</th></tr>
<tr><td colspan="2"><i>1. History of the Case.</i></td></tr>
<tr><td>Usually preceded by a thoracic affection of longer or shorter duration, an injury to the air-passages, or a predisposition to phthisis.</td><td>Preceded by disease of the stomach or liver, by poisoning, etc. (see Etiology).</td></tr>
<tr><td colspan="2"><i>2. Mode of Attack.</i></td></tr>
<tr><td>The attack begins with constriction in the chest, tickling in the throat, and cough. Afterwards choking, and vomiting of the swallowed blood may also occur.</td><td>The act of hæmatemesis begins with a sensation of fulness in the epigastrium, nausea, and inclination to vomit. During the attack cough may be excited from irritation of the larynx by blood, which has flowed into this cavity.</td></tr>
</table>

3. *Character of the Blood Discharged.*

The blood is *bright-red*—at least during the first part of the attack—*frothy* from admixture with air in the bronchi, and of an *alkaline* reaction. It also contains bronchial secretion, mucus, and sometimes pus.	The blood is usually *dark* in color, but it may be bright-red if the hemorrhage be considerable; it is *clotted*, contains no air bubbles, and under some circumstances, particularly if the ingesta are discharged in large quantity at the same time, it has an *acid* reaction. It is often mixed with food in different stages of digestion.

4. *Concomitant Symptoms.*

On examination of the thoracic organs, abnormalities in the percussion, or at least in the auscultatory, signs are discovered; moist râles limited to a particular spot. Epigastrium normal on percussion. More or less marked cardiac or pulmonary disease.	No moist râles limited to a particular portion of the lung. In cases of considerable extravasation of blood into the cavity of the stomach, the organ is dull on percussion. Marked symptoms of disease of the stomach, or liver, etc.

5. *Symptoms following the Attack.*

Moist râles continue in a limited portion of the chest. *The sputa still remain bloody for several days. Fever* quite common, due to the fundamental disease.	Stools colored black. *Sputa perhaps at first bloody, but become clear immediately after the attack. Fever only in exceptional cases,* and then occasioned by the disease which induced the hæmatemesis.

Besides the acid reaction of the discharged blood, which, however, occurs only in certain cases; I hold *the most important indication in favor of hæmatemesis to be the absence of bloody sputa discharged with coughing.* For although in hæmatemesis blood may be coughed up during the attack itself, yet, considering the mode of origin of this bloody expectoration, it is hardly possible for the latter to continue for several days; while in hæmoptysis this is commonly the case, the blood disappearing from the sputa very gradually.

In many cases, however, the differential diagnosis still remains difficult. Thus I remember at Griesinger's Clinique a case in which a hemorrhage took place in the presence of the house physician, who regarded it as unquestionably an hæmoptysis, whereas after death the condition of the stomach left no doubt that the attack had been one of hæmatemesis.

Whether the bleeding is of gastric or of intestinal origin can usually be readily determined if the blood be discharged by *vomiting*, because it is only in extremely rare cases (*e. g.*, in duodenal ulceration) that blood regurgitates from the intestine into the stomach, and therefore the chances in a hæmatemesis are overwhelmingly in favor of the former alternative. When, however, the blood is discharged only by stool the differential diagnosis is difficult, and can be made only from the other symptoms of disease, since the blood, whether coming from the stomach or from the upper part of the intestine, is exposed about an equal length of time in the digestive canal, and is always discharged in the form of a tarry substance mixed with the fæces.

The gastric origin of the vomited blood having once been determined, the next question to be decided is as to the cause of the hemorrhage. In the great majority of cases this question will present no important difficulties, if we keep in mind the points laid down in the chapter on Etiology, while the same rules will enable us to determine, also, whether the hemorrhage proceeds from large vessels or from capillaries. In cases of "internal hemorrhage" death usually ensues so rapidly that we are obliged to be content with a brief physical examination and a hurried survey of the possible causes of the accident.

The existence of *hemorrhagic erosions* in the stomach cannot be diagnosticated in the majority of cases during life, although their presence may always be expected in morbid conditions which are attended by stases of the gastric vessels and by gastric catarrh. When under such circumstances the vomited matters contain a scanty amount of blood, and the other causes of gastric hemorrhage, such as carcinoma, ulcer of the stomach, direct injuries, etc., can be excluded, we may suspect that, along with the hemorrhage upon the free surface, extravasations of blood have occurred also into the tissue of the wall of the stomach. A *certain* diagnosis of this affection, which possesses rather a pathologico-anatomical than a clinical interest, is entirely out of the question.

Prognosis.

While in many cases of disease of the stomach the presence of blood in small quantities in the vomited matters, especially if it occur frequently, is an important indication—as, for instance, when carcinoma ventriculi is suspected—yet its significance should not be over-estimated. I have often noticed, when severe choking movements have been excited by the introduction of the stomach tube, that the contents of the stomach which were washed out contained points or even large streaks of blood without the latter having any diagnostic or prognostic significance whatever. They are met with even when the stomach of a healthy person is washed out for experimental purposes. I have seen these traces of blood in the washings of the stomach so frequently that they have ceased to alarm me; I have never seen any bad results from them, although, of course, I have recommended rest, and, when possible, entire abstinence from food on the following day.

Even larger hemorrhages from the vessels of the stomach are of themselves not so dangerous as one would suppose from theoretical considerations and from the alarming impression which every copious "gush of blood" involuntarily makes. A comparatively large number of patients with hæmatemesis recover from the most hopeless anæmic states, and we should therefore not despair of saving the patient until death has actually taken place. The reason why the progressing weakness, which, under other circumstances, is so dangerous a symptom, is less to be feared in hæmatemesis, lies in the fact that the diminished force of the heart's action, and the increasing pressure upon the wall of the stomach effected by the extravasated blood, both favor coagulation at the bleeding point. Many hemorrhages—*e. g.*, those which occur in cirrhosis of the liver—even exert in a certain sense a favorable influence, by depleting the distended capillaries, in this way not only temporarily relieving the gastric catarrh which depends upon the congestion, but perhaps also producing absorption of the ascites. Still the loss of blood is, under all circumstances, a depressing factor, and it should never be forgotten that one of the chief dangers in hæmatemesis arises from the tendency of the fundamental disease to induced recurrences of the attack. Hemorrhages produced by the erosion of large vessels are of course specially dangerous; while the vicarious hemorrhages, which depend upon simple capillary congestion, are usually of a harmless character.

Treatment.

If we were able in any way to act upon the *causes* of gastric hemorrhage, we might at least diminish the severity of the attack. But, unfortunately, it is only in very exceptional cases that such a mode of treatment is possible, as a glance at the etiology of gastric hemorrhage must sufficiently show. Still, opportunities for prophylaxis occasionally arise; thus when the bleeding is occasioned by poisoning, antidotes should be given to prevent further erosions and denudation of the blood-vessels; in the regularly recurring gastric hemorrhages, which take place at the normal menstruation, leeches may be applied to the cervix uteri at the time when the attack is expected to occur; while in persons who suffer from attacks of hæmatemesis, resulting from cirrhosis of the liver, blood may be withdrawn from time to time from the hemorrhoidal veins. So also the tendency to hemorrhage from the gastric vessels in chlorosis should be combated by ferruginous preparations, and in scorbutus by improving the hygienic conditions of the patient.

When hemorrhage has once occurred, the first indication is to avoid everything which can increase the force of the heart's action, and thus heighten the blood pressure in the vessels of the stomach. With this object the most perfect *rest* in every respect is to be insisted upon; the patient should lie down, and should change his position as little as possible; the intense alarm, which is usually manifested by the patient on the occurrence of the hæmatemesis, should be allayed by a few reassuring words, and his attendants requested to maintain absolute quiet. The bed-chamber should be kept cool and the bed-covering light, in order not to increase the uneasiness of the patient by interfering with the respiration. Secondly, care should be taken to avoid any local irritation of the bleeding mucous membrane of the stomach by hot, highly seasoned food, or by coarse articles of diet which inflict mechanical injury, etc. The best plan is to withhold food by the stomach for some days, or even for weeks, if one wishes to be very cautious, and to nourish the patient by the meat-pancreas injections, in order to avoid not only all irritation but also all unnecessary contractions of the wall of the stomach, whereby the recently formed thrombi might be dislodged.

The various causes which interfere with the arrest of the hemorrhage being thus as far as possible removed, the question now arises whether we have any remedies which directly control dangerous hemorrhage, and, if so, which of them promises to be the most effectual. This question is all the more important, because, in our alarm at the gravity of the situation, we are only too apt to ply the stomach with one "styptic" after another without having convinced ourselves of the real effectiveness of these remedies.

In view of the common experience that even the most severe hemorrhages often cease spontaneously when a considerable degree of anæmia has been induced, it is not at all surprising that in former times *venesection* was recommended as a remedy for hæmatemesis. But, of course, the mere continuance of the hemorrhage will of itself accomplish this result, without any venesection, by diminishing the force of the heart's action and the pressure of the blood; moreover, it is scarcely necessary to point out how dangerous such an artificial abstraction of blood must be in those cases where a little more or a little less blood lost makes all the difference between a complete arrest of the heart's action, and a diminution of its force just sufficient to stanch the bleeding. From this point of view, also, the *application of irritants to the skin*, not only those which produce a very transient, but also those which produce a more continued retention of blood in the skin, *e. g.*, large dry cups, are, to say the least, of doubtful expediency; less objectionable are mustard poultices and mustard foot-baths, followed, if necessary, by cold compresses; or, also, the practice extolled by the ancients, of ligating the extremities for the purpose of preventing the reflux of the venous blood in the surface.

For the purpose in view—viz., the reduction of the blood pressure—these measures, which act either by abstracting blood or by altering its distribution, are not so well adapted as remedies which act upon the heart and the conditions of blood pressure in a gradual and direct manner, such as muscarine, digitaline, etc. But, aside from the fact that we have no right to experiment with these poisons upon individuals who have nearly bled to death, at all events not in the present condition of our knowledge of their action, I have myself never ventured to use the favorite remedy for arresting hemorrhage—digitalis—because I have feared equally its power in small doses to increase the blood pressure, and in larger ones to excite vomiting. My experience with *ergotin*, however, as regards its hæmostatic action would lead me to recommend it, although I am aware that physiological experiment has thus far entirely failed to explain its mode of action. Still, the very recent experiments of Hermann[1] seem to show that ergotin *diminishes* the blood pressure. I use it dissolved in water one part to ten, and inject it hypodermically in doses of fifteen drops at a time, repeating the injection several times if necessary.

Still more open to criticism than the employment of these remedies is the administration of the *styptics* proper. What surgeon has not smiled at seeing us use the chloride of iron in hæmatemesis in a dose (usually from six to ten drops of the solution in a glass of water), which must seem to him ridiculously small in view of his own failures to obtain a styptic effect even with the pure article? Moreover, this dilute solution usually passes into a stomach filled with blood and partly digested food, and thus fails to come into contact with the surface of the mucous membrane. The same fact should be borne in mind, also, in giving *acetate of lead*, *tannin*, *alum*, etc., in order that too much may not be expected from the use of these hæmostatics, which have acquired their reputation as styptics, partly on account of their directly precipitating the fibrin of the blood, and partly on account of their astringent effect upon the gastric mucous membrane. Acetate of lead is given in doses of from three-quarters to one and a half grains, tannin up to five grains, while the best form of administering alum is the alum whey, drank by the tumblerful in small quantities at a time; all these styptics unfortunately excite vomiting when given in large doses. I ordinarily use the alum whey, which is usually well borne by the patient.

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[1] Handbuch der Experimentellen Toxicologie. 1874. S. 387.

Another hæmostatic, which is more efficacious than any yet mentioned, is *cold.* The effects of this agent are best obtained by reducing the temperature of the contents of the stomach with the use of *ice pills*, in connection with the external application of ice to the epigastrium, although, as regards the latter method, it is doubtful whether the penetrating effects of the cold are sufficient to be of practical benefit. According to the experiments of F. Schultze,[1] an ice-bladder laid upon the epigastrium may be expected to reduce the temperature of the anterior wall of the stomach only by from 2° to 4° F., and that of the posterior wall by less than ½°. Another advantage which the ice-pills possess over other styptics, is that the former may be used even when the stomach is filled with blood, while the latter quickly lose their effect in such cases by their still further dilution in the fluids of the stomach.

The special treatment of certain alarming or annoying symptoms, which occur during the course of gastric hemorrhage, still requires to be mentioned.

The *attacks of syncope* are to be treated by rest in the *horizontal* posture, for the purpose of favoring the flow of blood to the brain. The use of restoratives, which excite the heart to greater activity, should not be resorted to at too early a period, because, as we have seen, the feebleness of the heart's action is one of the factors in the formation of the life-preserving clot. When the syncope is profound and dangerous, the most powerful restorative is ether injected hypodermically, several syringefuls being used one after the other. The distinct odor of ether in the breath will show that the remedy has been absorbed. Ether is preferable to other stimulants,[2] because it can be introduced into the body in this way without coming in contact with the diseased organ. Other restoratives, such as camphor and the spirits of ether, with the anisated solution of ammonia (Zülser), may also be injected hypodermically. Water of ammonia held before the nose, sprinkling the face with cold water, etc., will in many cases be sufficient to revive the patient. Mention has already been made of the danger in very feeble persons from the impaction of clots of blood in the entrance to the larynx ; of course, when suffocation impends from this cause, the clots should be immediately removed.

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[1] Deutsches Archiv für klin. Medicin. XIII. S. 500.

[2] Alcohol has been repeatedly used in the same way with good results.—*Tr.*

The excessive *thirst* is best relieved by swallowing pieces of ice, which also diminish the *tendency to vomiting.* If the latter symptom be very obstinate, sinapisms may be used ; as for injections of morphine, I am afraid of them, because physiological experiments have shown that the blood pressure begins to increase under their use, and for this reason I would not recommend them, while the patient is under mental and vascular excitement, until more harmless measures have proved ineffectual.

The *after-treatment* consists in guarding, as far as possible, against recurrences of the hemorrhage. For some time, therefore, after the attack it is advisable to spare the stomach by using only beef tea, or similar easily digestible articles of food, and to return to the usual diet very gradually. The administration of emetics and cathartics, after the cessation of the hemorrhage, for the purpose of ridding the stomach of the remaining blood, has become antiquated. The best protection against the recurrence of the attack is the treatment of the fundamental disease upon which the hæmatemesis depends.

The therapeutics of gastric hemorrhage should not be dismissed without considering the question, whether, in cases of impending death from this cause, the introduction of blood by *transfusion* is allowable, or is in fact demanded. The number of cases in which this operation has been performed under these circumstances is too small to permit a positive decision in regard to its general applicability. Although the alarming anæmia, which ensues after severe hemorrhages from the gastric vessels, seems to indicate clearly the transfusion of new blood into the body, yet a more careful consideration will show that the operation is unquestionably attended by great danger to the patient, arising from the heightened pressure produced in the vascular system, and the danger of, in this way, exciting another and fatal hemorrhage by the dislodgement of the thrombus. That this danger from transfusion is not merely theoretical, but actual, is shown by a case reported by Kussmaul and Czerny,[1] in which the operation, performed shortly after a profuse intestinal hemorrhage, and indicated by the vital conditions, was followed by the discharge of large quantities of bright red blood, profound anæmia, collapse, and death. The operation should, therefore, at all events, be deferred until the last moment, and the blood should be injected in very small quantities at a time. Of course, when the operation is proposed for the relief of dangerous anæmia several days after the hemorrhage has ceased, the case is different. Here the danger suggested is entirely subordinate, and the operation ranks as the most effectual remedy against the pernicious effects of loss of blood.

Neuroses of the Stomach.

Under this head—*the neuroses of the stomach*—are to be enumerated all those morbid conditions in which *the derangements are essentially limited to the nervous apparatus of the stomach.* This definition is no doubt incomplete, but not more so than our knowledge of normal functions of the gastric nerves, nor than our knowledge of the entire clinical character of neuroses of the stomach. Formerly, when autopsies were rarely made, and pathological anatomy was still undeveloped, this class of diseases, the diagnosis of which is based almost exclusively upon the existence of subjective symptoms, must naturally have been still more comprehensive than it is now. Not until clinical observation could be corrected in the light of post-mortem observations, was it possible to eliminate from the class of neuroses those cases of "cramp of the stomach," "pain in the stomach," etc., in which palpable changes in the structure of the organ were found after death, and in which, therefore, the functional derangement of the nerves was to be regarded simply as the expression of a pathologico-anatomical lesion, such as cancer of the stomach, gastric ulcer, etc. Notwithstanding the most industrious macroscopic and microscopic investigations by anatomists, clinical physicians still meet with cases in which these nervous symptoms occur without any perceptible alteration in the tissues of the stomach. Probably in these cases, also, the functional derangements depend upon anatomical changes, and quite likely upon changes in the gastric nerves. Hitherto, however, only gross lesions in the track of the gastric nerves have been discovered; thus, for example, in a case recorded by Joseph Frank (Præc. Vol. II., pars. II., 2. p. 292), in which vomiting, nausea, and distress in the stomach had continued for two years, the thoracic vagus was found after death imbedded in a "steatomatous" tumor; but in by far the larger proportion of neuroses nothing abnormal has been observed in the nerve tissue. It will be more correct, therefore, at present, to give a rather negative wording to our definition of gastric neuroses, and to include under it those affections which *relate particularly to the functional derangements of the stomach, and in which nothing is to be found, in an anatomical respect, which is opposed to the supposition that the disease is situated exclusively in the nervous system.*

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[1] *Berns*, Beiträge zur Transfusionslehre. Freiburg. 1874. S. 27.

If the normal functions of the nerves of the stomach, as manifested by movements, by secretion, and under certain circumstances, by sensibility, be taken as the basis for a classification of the various gastric affections, which are traceable to altered nerve-function, the following varieties of neuroses will be obtained according to the predominance of an increased or of a diminished irritability of the gastric nerves:

Increase or diminution of sensibility.
Increase or diminution of contractility.
Increase or diminution in the amount of secretion.

All these forms of neuroses are illustrated in the pathology of the stomach; at least we have evidence that they occur clinically. They also undoubtedly combine with each other, for example, hyperæsthesia with hyperkinesis, as is shown by cases in which the excessive sensitive irritation of the gastric nerves results in a reflex spasmodic contraction of the muscular coat of the stomach, the gastralgia being accompanied by visible vermicular movements in the epigastrium ("cramp of the stomach").

Our knowledge in regard to the conditions and the mode of activity of the gastric nerves is so very meagre that it is impracticable at present to give a clinical picture of each of these forms of neuroses. We shall, therefore, describe here only the variety with which we are most familiar under the term "gastralgia;" the other forms of nervous derangement are either subordinate symptoms of a general neurosis, such as hysteria, or they enter so largely as anomalies of secretion into the production of dyspepsia and chronic gastric catarrh that they have already been considered at length in a previous chapter.

Gastralgia.

Syn.: Magenkrampf, Gastrodynie, Cardialgie, Erethismus ventric., Hyperæsthesia ventriculi.

Musgrave, De arthritide symptomatica. 1736.—*L. Heister*, Medicinische chir. u. anat. Wahrnehmungen. 1753. Wahrnehmung. No. 372. S. 614.—*Odier*, Sammlung auserlesener Abhandlungen. XII. 826 (1786); the first recommendation of bismuth for gastralgia.—*X. Müller*, De usu argenti nitrici. præs. *Autenrieth*, Diss. Inaug. Tübingen. 1829.—*Hornung*, Affectiones ventriculi arthriticae. Oesterr. medic. Jahrbücher. 1836. S. 401.—*Weitenweber*, Therapeutische Abhandlg. über den Kaffe. Oesterr. Jahrb. 1846. X. 1. Abs. IV.—*Ritter*, Zur Pathologie der Cardialgie. Heidelberger Annalen. 1848. Ref. Schmidt's Jahrb. 59. 180.—*Möller*, Ueber eine eigenthümliche Form von Hyperästhesie des Magens bei Anämischen. Deutsche Klinik. No. 32. 1851.—*Putegnat*, Ueber perniciöse Wechelsfieber. Union. 80–82. 1853. Schmidt's Jahrb. 80. 317.—*Fenger*, Praktische Bemerkungen über die Cardialgie und deren Behandlung. Referat aus dem Dänischen. Schmidt's Jahrb. 97. 317.—*v. Franque*, Bericht aus der Münchener Poliklinik. Deutsche Klinik. 1856. S. 241.—*Schramm*, Ueber Cardialgie. Bayer. Intelligenzblatt. 29 u. 30. 1860.—*Garrod*, The nature and treatment of gout, etc. 1873.—*Massart*, Traitement des congestions, etc. par la médication arsenicale. Gaz. hebd. 1863. pag. 179, with an historical account of the use of arsenic.—*Day, W. H.*, On Gastrodynia. Lancet. 1867. Dec. 21.—*Beard* and *Rockwell*, The medical and surgical uses of electricity. 1874.—In this bibliography numerous other works on the treatment of gastralgia are omitted, for the reason that they merely contain records of cases, and are without any special value for the development of the rational therapeusis of this disease. Only those works are mentioned which contain the first recommendation of one of the remedies in common use, or the literature in regard to the action and employment of the same.

Under *gastralgia* we include those morbid conditions of the stomach *which are limited essentially to the sensitive sphere of the gastric nerves; which manifest themselves in an excessive reaction of the same to certain irritants; and in which there are no anatomical reasons for not referring the symptoms entirely to disease of the nervous system.*

Some writers have asserted that the two nerves supplying the stomach—the *vagus* and the *sympathetic*—give rise to different forms of neuralgia, which they have accordingly distinguished as *gastrodynia neuralgica*, and *neuralgia cœliaca.* In the present state of uncertainty, however, with regard to the mode of action of the gastric nerves, especially with regard to the conditions of sensation, and in view of the anastomotic connections between the vagus and the sympathetic in the stomach, such a division of cardialgia appears to be both theoretically and practically untenable.

Etiology.

With this limitation to the conception of gastralgia, we shall naturally have to seek the causes of the affections in two directions, *either in an abnormal nature of the irritations to which the gastric nerves are subjected, or in an altered condition of the nerves themselves, which therefore react abnormally with a normal degree of irritation.* Of these two modes of genesis the former is by far the most frequent, but in many cases the two causes are doubtless associated with each other; thus the unusual or abnormally severe irritation may excite a gastralgia, or at least one of *such violence*, only because the gastric nerves are more sensitive or are more intensely irritated than usual.

Abnormal Character of the Irritants, which Act upon the Gastric Nerves, as a Cause of Gastralgia.

Under this head belong, in the first place, physical or chemical changes in the *ingesta*. Thus cardialgia may be produced by an immoderate indulgence in food, by an excessive accumulation of gas, by worms which have passed into the stomach, by very cold or very hot food, by sharp bodies, such as fruit seeds, etc., or by fermenting, acid, or otherwise chemically changed ingesta. English writers maintain, also, that the abuse of tea and coffee has a marked tendency to produce the same result. In certain cases, from an idiosyncrasy of the individual, the sensitive nerves of the gastric mucous membrane are excessively irritated by particular articles of food, which are tolerated without difficulty by other persons.

While as a rule the irritation of the sensitive gastric nerves is peripheral—that is to say, proceeds from the mucous membrane of the stomach—the irritation may, under other circumstances, originate in the nerve centres, and in accordance with the law by which sensations of centric origin are transferred to the periphery of the nerves may be felt as a gastric pain. In this way cardialgia is reported to be produced by the pressure of tumors upon the vagus, and by diseases of the brain and spinal cord. Well-marked gastralgia, depending upon cerebral or spinal disease, (excluding those cases in which an intercostal neuralgia of spinal origin localizes itself in the epigastrium), if it ever occur, is at all events extremely rare, although Krukenberg mentions pain in the stomach as a frequent accompaniment of diseases of the brain and spinal cord.

More frequent are cardialgias resulting from irritations, which, transmitted from other viscera along the track of the sympathetic, excite associated sensations in the gastric nerves; *e. g.*, gastralgia in diseases of the intestines, bladder, etc., and especially in affections of the female genital organs. The connection between the latter affections and the occurrence of gastralgia is shown by undoubted clinical evidence, for instance, by the striking case of Niemeyer's,[1] in which cardialgic attacks recurred every four weeks in the place of the menses, and could be excited artificially at any time by the application of leeches to the neck of the uterus. Besides amenorrhœa, the patient was also suffering from retroflexion of the uterus and erosions of the cervix.

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[1] Lehrb. der spec. Pathol. etc. Aufl. 8. S. 591.

Abnormal Conditions of the Gastric Nerves as a Cause of Gastralgia.

The most frequent causes of gastralgia are *hysteria* and *chlorosis*, two affections, therefore, which occur almost exclusively in the female sex.

The association between cardialgia and *chlorosis* is particularly frequent. While it cannot be denied that the cardialgia in many of these cases is not a pure neuralgia, but is merely a symptom of the gastric ulcer, which frequently accompanies this affection, it is equally certain that in numerous instances the cardialgia is solely dependent upon the chlorosis. In fact, it seems to me that much harm has been done of late in these cases by our too ready resort to the diagnosis of "gastric ulcer." I am convinced, by my own experience, that young girls frequently suffer from the milder forms of gastralgia, which are badly treated, because we are afraid of diagnosticating the case as one of simple gastralgia. Upon this point I shall give some evidence hereafter. The question as to what is the nature of the causal relation between chlorosis and gastralgia can be answered only hypothetically. In view, however, of the well-established fact that changes in the normal nutrition of the nerves produce a disturbing effect upon the irritability of the latter, it is certainly not surprising that neuralgias should occur in an affection like chlorosis, in which a general mal-nutrition of the body must be the necessary result of the altered composition of the blood. During convalescence from exhausting diseases, during the course of tuberculosis and of various other affections, in which the nutrition is impaired, gastralgia may also occur as an expression of depressed vitality.

The perverse irritability of the entire nervous system, which characterizes *hysteria*, and which is usually regarded as depending upon a general mal-nutrition of the nervous apparatus, evidently extends in some cases to the nerves of the stomach; in fact, cardialgia is one of the most common symptoms of this affection, which, moreover, as is well known, is frequently associated with disorders of the female organs of generation.

In this connection should be mentioned, also, the gastralgia which occurs in *debauchees* and *onanists*. In many of these cases the neuralgia is no doubt due to the impairment of nutrition induced by masturbation, but in others it results directly from the masturbation without the intervention of the factor referred to.

I recently examined a young man with well-marked gastralgia, but failed, on the most careful inquiry, to discover its cause, as the patient was a hearty workman, who had returned from the campaign, and was entirely free from dyspeptic symptoms. I diagnosticated the case, however, as one of gastralgia, and put him upon the proper treatment. After a time, however, the patient confessed to venereal excesses and extremely frequent pollutions.

In such cases we must suppose that sensational sympathies exist between the nerves of the genital organs and those of the stomach in men similar to those which we have been compelled, for clinical reasons, to infer for the genital sphere of women.

The attacks of gastralgia which are observed in *arthritis* may also be referred to mal-nutrition of the nervous system. Musgrave, even as far back as 1736, gives an account of arthritic cardialgia; Romberg also enumerates arthritis as one of the causes of this affection—in fact, an attack of arthritis in his own person was ushered in by a severe "neuralgia cœliaca."

Still another toxic cause of gastralgia is *malaria*. In some of these cases the gastralgia seems to me, from the description, to be merely a symptom of intermittent fever, like the headache, the oppression, etc., while in other cases it is evidently a masked form of the malarial paroxysm.

In accordance with the definition of gastralgia given above, our classification will necessarily have to include those cases of pain in the stomach which follow the *healing* of ulcers, and which appear to be due to the compression of a nerve-twig by the cicatrix. On the other hand, the cardialgia in open ulcers, in cancer, etc., ought to be regarded merely as a symptom of these affections, under which heads it has already been considered.

Finally, it is not to be disguised that in certain cases of marked gastralgia none of the causes previously mentioned adequately explain the origin of the neuralgia.

Gastralgia is a disease chiefly of adolescence and middle life, and while, as we have seen, it occurs in both sexes, women are probably more subject to it than men. In many regions it is particularly common—for instance in Sweden, according to Huss and Fenger, where it is said to be due to the excessive use of spirits and coffee; also in malarial districts, where it may occur as a masked form of intermittent fever.

Symptomatology.

The severe paroxysmal pain, which is the characteristic feature of gastralgia, occurs either suddenly without premonitory disturbance, or after being preceded by weight in the epigastrium, salivation, nausea, headache, and other nervous symptoms. In severe paroxysms the patient is attacked by a griping, tearing pain in the epigastrium with a sensation of impending death, as if "claws were clutching the pit of the stomach," as Romberg expresses it in his brief and striking account, which I have adopted as the basis of my own description. This pain radiates from the point of the ensiform process into the surrounding parts as far as the back, and increases in severity until it sometimes produces faintness and convulsions. At the same time the epigastrium is generally altered in shape, being either distended into a globular form, or retracted against the spine; the extremities are cool, the face sunken, and the pulse small and intermittent.

The concomitant symptoms are vomiting, sometimes occurring at the beginning, sometimes at the termination of the attack, and a great variety of sympathetic sensations and reflex movements, such as clavus, globus, urgent micturition, craving for food, spinal irritations (Bamberger), eructations, yawning, contractions of the abdominal muscles, etc.

Another point of some diagnostic importance is the fact that pressure upon the epigastrium is not only borne without increase of the pain, but is in fact resorted to by the patient himself, either by pressing the epigastrium against some solid object, or by bending himself together, or by lying on his stomach. Some patients also take food during the attack for the purpose of satisfying the distressing craving for food. Such methods of relieving the pain are of course not practised by all patients.

The duration of the attack varies considerably; sometimes it lasts a few minutes, sometimes several hours, and subsides gradually, with eructations, vomiting, the copious discharge of a clear urine, which in hysterical patients is strikingly pale (urina spastica), and, finally, with great exhaustion.

As in other forms of neuralgia, free intervals alternate with the attacks of severe pain. For this reason the nutrition of the patient is not materially impaired, provided the fundamental disease, upon which the neuralgia depends, is not itself of an exhausting character. Besides the cardialgic attacks, symptoms of derangements in other organs may also be present, varying according to the disease which produces the neuralgia, such as menstrual disorders, migraine, globus, etc., in hysterical patients, or shortness of breath, palpitation of the heart and supraorbital neuralgia as results of chlorosis, etc. A typical occurrence of the paroxysms with perfectly regular intervals is noticed in the gastralgia which accompanies uterine disease, particularly disorders of menstruation, and also in the gastrodynia which depends upon malarial infection.

From these severe forms of gastralgia I wish to distinguish a less serious *general hyperæsthesia of the gastric mucous membrane*, which is most apt to be met with in chlorotic patients, especially those of the female sex, and which is attended by anorexia, weight and pains in the epigastrium. My own experience with this form of neuralgia has been insufficient to enable me to give a distinct portraiture of the affection, and I shall, therefore, content myself by referring to what I have already said upon this point in the chapters on the diagnosis of chronic gastric catarrh and ulcer of the stomach. I am satisfied that in doubtful cases a diagnosis of "gastric catarrh" or "gastric ulcer" is made too frequently, as is shown by the entire failure of dietetic and medicinal treatment directed against these affections, and the success of treatment by electricity and a more generous diet.[1]

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[1] Möller also some time since pointed out the occurrence of such forms of disease

The *course* of the disease is *chronic.* An exception to this rule is formed by those cardialgic attacks, which are produced by abnormal ingesta, *e. g.*, worms, etc., and which usually at once disappear after the removal of the *materia peccans.*

Diagnosis.

In the diagnosis of gastralgia the main point in the correct interpretation of the disease lies in a careful exclusion of the other affections for which it may be mistaken. The pains which constitute the most important symptom of this disease play so prominent a part in the complaints of the patient, that we are not likely in doubtful cases to overlook the possibility of a gastralgia. On the contrary, the real difficulty in many cases is to avoid being unduly influenced by the incessant complaints of cramp and pain in the stomach, so that the detection of the morbid conditions which are the real cause of the epigastric pain, requires a thorough and unbiassed investigation.

Pains in the epigastrium, originating in *rheumatism of the abdominal muscles*, are sometimes referred by patients to the stomach; but closer examination will easily show that these sensations are located in the abdominal wall. On the other hand, the distinction between gastralgia and *neuralgia of the inferior intercostal nerves*, which are distributed to the epigastrium, is more difficult. If it be borne in mind that this form of neuralgia is especially apt to occur in chlorotic persons, and that these patients are particularly liable also to digestive disorders, we can readily see how such a combination of dyspepsia, with an intercostal neuralgia limited to the epigastrium, may be mistaken for gastralgia. This error in diagnosis may be avoided by palpation of the affected intercostal spaces, where a painful point will here and there be discovered; also by electrization of the intercostal nerves in the back—a procedure which may clear up the case by causing the epigastric pain to disappear.

So, also, when painful sensations originating in certain abdom-

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which he thought ought rightly to be regarded as neuralgic, because in some of his cases the gastric affection distinctly alternated with other forms of neuralgia.

inal organs—the liver, pancreas, uterus, kidneys, etc.—radiate into the epigastrium, a similar mistake in diagnosis may be committed unless the starting-point of the pain and the other attendant symptoms are carefully observed. The most frequent source of error in such cases is the *colic resulting from biliary calculi*—a mistake all the more pardonable as a distinction between the two conditions is at first scarcely possible, when the pain is located exactly in the epigastrium, the liver is apparently neither painful nor enlarged, the gall-bladder cannot be felt, and no icterus is present. The differential diagnosis between simple gastralgia and pains originating in the organs mentioned sometimes presents special difficulties from the further fact that, as already remarked, sympathetic gastralgic attacks are in these cases of extremely frequent occurrence. It is always necessary, therefore, even in clear cases of gastralgia, to examine also for other remote sources of pain before deciding that the gastralgia is the sole disease. The distinction between gastralgia and the gastralgic attacks which depend upon *ulcer of the stomach* has already been considered under the head of the latter affection. In doubtful cases—and these, as was fully shown in that article, are by no means rare—it is advisable to treat the patient as if he were suffering from ulcer of the stomach, in accordance with the rules there laid down. If the pain be not materially mitigated by this treatment, but yield to the use of the constant current, a tolerably certain diagnosis may thus be made *ex juvantibus et non juvantibus*, and the further treatment be modified accordingly. At all events, this mode of proceeding can do no harm, and may perhaps spare us the great regret of seeing the supposed gastralgia suddenly revealed as a gastric ulcer by the occurrence of a fatal hæmatemesis.

Cancer of the stomach and *chronic gastric catarrh with cardialgic attacks*, are less likely to be mistaken for simple gastralgia. The occurrence of the former disease in old persons, the coincident cachexia, and the characteristic vomited matters generally point unmistakably to cancer, even when no tumor can be felt. Gastric catarrh may also be usually distinguished by the more or less uniform continuance of the pains, which are generally very moderate in severity, by the dyspepsia which is directly connected with taking food, and by the absence of all typical symptoms during the course of the disease.

Although in the great majority of cases it will be possible, with these diagnostic aids, to recognize a gastralgia with certainty, yet in other instances the existence of a pure gastralgia will still remain doubtful—a result, however, which is the less objectionable, either for the physician or the patient, because it will lead the former to further careful examination, and the latter to caution in diet.

But even after the diagnosis of gastralgia has been placed beyond all doubt, we ought not to stop here in our inquiry into the nature of the disease, because it should never be forgotten that the neuralgia of the gastric nerves is always, in fact, merely the expression of another affection, the exploration of which is a still more important task for the physician than the recognition of the gastralgia itself. For no permanent benefit can be expected from treatment until the disease is attacked radically, and until the occasion for the occurrence of new attacks is withdrawn by the cure of the fundamental disease.

Prognosis.

Notwithstanding the serious aspect which gastralgia sometimes assumes, the prognosis, *quoad vitam*, is favorable, since the paroxysm is never fatal, provided the gastralgia is unconnected with structural changes in the stomach.[1] On the other hand, the prospect of a speedy, complete, and permanent cure is not very favorable, and will, of course, depend entirely upon the cause which excites the attacks. Thus, when the gastralgia is produced by unusual articles of food which can be immediately removed from the stomach, there is more hope of a rapid and permanent cure than when the disease depends upon anæmia or hysteria. Still, even in the latter affection, the prognosis is not very unfavorable if the hysteria be found to depend upon causes which are within the reach of treatment. Moreover, hysterical cardialgia often disappears with the advance of life.

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[1] Heister's case, which is often cited on this point, of a woman, thirty-three years of age, who died in 1775, with gastric pains of three days' duration, and convulsions, cannot be adduced as evidence of the possibility of a simple gastralgia terminating fatally, for the reason that in this case poisoning could not be excluded, and, also, because "at the cardia, where worms were lodged, the stomach was bloody, and looked as if it were eroded and gangrenous."

The comparative prognosis in the different forms of gastralgia may be represented by the following series, beginning with the most curable: gastralgia induced by the retention of unusual articles of food in the stomach, then the forms produced by malaria, chlorosis, uterine disease, arthritis, onanism, hysteria, cicatrices in the stomach, and, finally, the gastralgia produced by cachectic conditions resulting from malignant, wasting diseases (tuberculosis, etc.). The prognosis is bad, also, when no cause can be discovered after the most careful examination, because such cases afford no indications for treatment.

Treatment.

In this affection, in which we have an unusually sure etiological basis for treatment, the carrying out of the causal indication is the most important task for the physician. Fortunately, in many cases we are able to meet this indication, and the treatment of the gastralgia will therefore frequently coincide with the treatment of the fundamental diseases which have given rise to it. A detailed consideration of the latter would be inappropriate here, and I shall, therefore, merely refer to certain leading points.

When the gastralgia is occasioned by the presence of *abnormal ingesta in the stomach*, their removal is obviously an indispensable part of the treatment. Unless special contra-indicacations are present (as, for instance, when sharp bodies of considerable size have been swallowed, whose removal would be attended by great difficulties), this may be best affected by the use of the stomach-pump, or by emetics, the most suitable of which is apomorphia (in doses of from a thirteenth to a sixth of a grain), because it can be used hypodermically without producing any injurious effect upon the gastric mucous membrane. If the washing out of the stomach, or the administration of an emetic, be unsuccessful, the attempt must be made to get rid of the noxious ingesta by the bowels. If necessary, the cathartic may be preceded by the administration of demulcents for the purpose of diminishing the irritating action of the ingesta upon the mucous membrane of the digestive tract.

The gastralgias which arise from *sympathy* with other diseased organs will most probably cease when we have succeeded in removing the original derangements, as is clearly shown by cases in which relief to a gastric neuralgia has been observed to follow the successful treatment of uterine disease. Thus, in Romberg's[1] case of a woman who was suffering from erosions on the os uteri, with anteversion of the uterus, and at the same time from cardialgic attacks, occurring chiefly at the periods of menstruation, these attacks, after having resisted every narcotic, disappeared entirely when the uterine disease was subjected to local treatment.

Since chlorosis or anæmia is so frequent a cause of gastralgia, it will often be found necessary to administer preparations of iron. The particular preparation which is to be used under such circumstances is not a matter of indifference, because the feeble stomach is more intolerant of some preparations than of others. I usually employ the lactate of iron in the form of powder, three grains at a dose, or reduced iron in three-grain doses, with equal parts of some aromatic powder or of extract of cinchona.

The imperfect digestion in chlorotic patients may also be benefited by the use of hydrochloric acid, eight drops to a wine-glassful of water two hours after eating.

In the case of *onanists* and similar patients, cold frictions of the whole body are advisable, or the patient may be taken away from his friends and his customary habits of life, and sent to the coast for sea-bathing, or to some elevated region, or, still better, to the ferruginous springs of St. Moritz, which, in my opinion, are admirably adapted to such invalids.

In *hysterical* gastralgias, the benefit to be derived from medicine is, as we all know, very precarious, so long as no cause, accessible to treatment, can be discovered for the hysteria.

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[1] Nervenkrankheiten. Aufl. 3. S. 158.

Even in the latter case, however, the attempt may be made to relieve the hysteria by the use of bromide of potassium, valerian, and other antispasmodics, by sea-bathing for the stronger patients, and by the judicious employment of hydropathy, etc., measures which need not be considered in detail here.

Gastralgias, which depend upon a *malarial affection*, require the bold use of quinine or arsenic; the latter is said to prove efficacious in many chronic cases when quinine has failed.

When the gastric pains are the result of *arthritis*, the usual treatment for gout is indicated, and particularly the use of large quantities of warm water. Moreover, since experience has shown[1] that the gout in the extremities disappears on the supervention of gastralgia, and, on the other hand, that the pains in the extremities return on its cessation, Garrod's recommendation, to attempt, by warmth and counter-irritants, to excite derivation to the joints, is certainly worthy of consideration; and all the more because the hot foot-baths, mustard poultices, etc., are so perfectly harmless that they can be used with the utmost freedom. At the same time Garrod uses stimulants, such as ether, ammonia, etc., when there is no co-existing inflammation of the stomach.

The *attack itself* may be treated by warm baths, mustard poultices over the epigastrium, in severe cases by inhalations of chloroform, and especially by the hypodermic injection of the *muriate of morphia*. For a century past *subnitrate of bismuth* has been highly commended, and has even been regarded as a true specific for gastralgia, like quinine for malaria. Others, as Massart, have obtained very striking results from *arsenic*, and have gone so far as to ascribe the effect of the bismuth to its contamination with this metal. In certain cases benefit has been derived from nux vomica, nitrate of silver, acetate of lead, coffee, etc. I have had no experience with any of the last-mentioned remedies, and, therefore, refrain from expressing any opinion in regard to them. Still, I believe that these drugs should not be resorted to until after a thorough trial of *electricity*, the one remedy for neuralgia which nowadays casts all others into the background. I have seen very good results from the use of the constant current—from ten to fifty elements, according to the sensitiveness of the patient, being necessary to produce a slight pricking sensation in the skin and to exert an alterative action upon the gastric nerves.

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[1] Garrod. 2. edit. p. 492, sq.

I place the anode upon the painful point in the epigastrium, and the cathode in the left axillary line, or further towards the spine, and continue the application for five or ten minutes. This rarely fails to diminish the pain, although sometimes only temporarily, and after a continuance of the galvanization for some time the pain may entirely disappear. In conclusion, an illustration of this treatment may be here given.

W., a foreman, forty years of age, and a vegetarian; formerly a dyspeptic, but for the past two years has been quite well. For the last three weeks he has suffered from a constant oppression in the epigastrium, alternating with pain which extends to the back, and almost entirely disappears when he lies down. When it is specially severe it is accompanied by gurgling in the epigastrium. At first the cardialgia did not begin until some hours after rising, and particularly until after some food had been taken. At present, eating seems to have no special influence upon the pain, and the appetite is unimpaired. There has never been any vomiting.

The examination revealed a very tender spot, as large as a silver dollar, in the epigastrium; general pressure over the whole region was painless.

I made a diagnosis of gastralgia, but did not conceal from myself that the change of the pain in different positions of the patient, the great sensitiveness of the epigastrium on localized pressure, the extension of the pain to the back, and the continuance of the painful sensations, made it at least questionable whether the cardialgic attacks were not rather symptomatic of a gastric ulcer, which was running a somewhat unusual course.

The indications pointed, therefore, to the use of the constant current for diagnostic purposes, as before described, and, if successful, to its therapeutic employment. On placing the positive pole of a constant battery, sufficiently strong to just excite pricking in the skin, over the painful spot in the epigastrium, the severe pain immediately disappeared. Every repetition of the galvanization produced the same effect, and after a few weeks the patient was cured.

Judging by the results which are also obtained by *faradisation* in the treatment of neuralgias, it is to be expected that this method will likewise prove useful in gastralgia. I have myself had no experience with it, because I have had no reason for giving up the treatment by the constant current, which, in the cases of gastralgia observed by me, has almost invariably proved successful.

Alterations in the Size, Form, and Position of the Stomach.

The most important of these conditions is dilatation of the stomach, the other alterations belonging to this category being of subordinate clinical importance.

Dilatation of the Stomach—Dilatatio Ventriculi—Gastrectasia.

A compilation of ancient and modern literature on this subject is to be found in *Penzoldt's* work on Dilatation of the Stomach, Erlangen. 1875. The following list of works includes only those of the most importance. In regard to ancient literature see also the treatise of *Joseph Frank*, referred to below.

Spigelius in *Rhodius*' Mantiss. anat. obs. XVIII. p. 13. 1623.—*Bonet*, Anat. lib. III. Sec. VIII. p. 807. 1679.—*Diemerbroeck*, Opera omnia. Lib. I. Cap. 7. 1685.—*Widmann*, Commerc. litt. ad rei med. et scient. natur. increm. Norimberg. 1743. p. 63.—*Gerh. van Swieten*, Comment. ad H. Boerhaave. Aphorism. etc. Comm. ad. aph. § 605. 10. 1754. Tom. II. p. 133.—*Mitterbacher*, Diss. de raro ventriculi casu, 1760, in *Klinkosch's* Sammlung Prager Dissert. No. 9. S. 159 (1771).—*Lieutaud*, Histor. anat. med. 1767. Lib I. Sec. II. obs. 21–25.—*Sauvages*, Nosologia methodica Classis. X. 18. 1. Meteorismus ventriculi. 1768.—*Morgagni*, De sedibus et causis morborum, etc. Epistol. XXXIX. 14–17. 1774. *J. P. Frank*, De curand. hom. morbis. Lib. V. pars. 6. § 666.—*Vetter*, Aphorism. aus der pathol. Anat. 1803. S. 169. ff. — *Klohss*, Merkwürdige letzte Krankheit des Hofrath Dr. Henning, in Hufeland's Journal der prakt. Heilkunde. 1824. S. 87. Stück 2.—*Piorry*, Die mittelbare Perkussion. übers. von Balling. Würzb. 1828. S. 261. ff.—*Duplay*, Archives générales, Nov. u. Dec. 1833.—*Pétrequin*, Bullet. de thérap. Tom. X. p. 239. Schmidt's Jahrb. 15. S. 36.—*Jos. Frank*, Prax. univers. praecept. p. III. Vol. I. Sec. 2. p. 225. Leipzig. 1835. This work contains copious references to the literature of the disease.—*Blumenthal*, Casper's Wochenschrift. 1835. No. 32.—*Pauli*, De ventriculi dilatatione diss. Frankf. 1839.—*Sérain*, Ref. Schmidt's Jahrb. Bd. 28. S. 137. 1840.—*Canstatt*, Jahresber. 1841.—*S. C.*, Enorme Vergrösserung des Magens. Gaz. di Mil. 1845. Schmidt's Jahrb. 51. S. 303.—*Oppolzer*, Klin. Vorträge, etc. Wiener klin. Wochenschrift. 1851. S. 305.—*Canstatt*, Spec. Pathol. u. Therap. 1856. Bd. III. S. 214.—*Traube*, Zur Lehre von der Magenerweiterung. 1861. Ges. Beiträge, etc. S. 986. 1871.—*Skjelderup*, Spontan. Ud vidurg, etc. Virchow's Jahresber. 1866. S. 134.—*Kussmaul*, Behandlung der Magenerweiterung durch eine neue

Methode mittelst der Magenpumpe. Deutsches Archiv für klin. Medicin. Band. VI. S. 455. ff. 1869.—*Ploss*, Deutsche Klinik. 1870. 8. 32.—*Jürgensen*, Deutsches Archiv f. klin. Medicin. VII. S. 239.—*L. Rosenthal*, Berliner klin. Wochenschr. 1870. Nov. 24.—*Popoff*, Berliner klin. Wochenschr. 1870. No. 38–40.—*H. Quincke*, Dilatat. ventric. Eigenthümliches Verhalten des Urins. Correspondenzbl. für Schweizer Aerzte. 1874. No. 1.—*Schultze*, Berliner klin. Wochenschr. 1874. No. 27. Brennbare Gase.—*Ewald*, Ueber Magengährung. Dubois-Reichert's Archiv. 1874. Heft 2.—*F. Penzoldt*, Die Magenerweiterung. Habilitationsschrift. Erlangen. 1875.

Pathological Anatomy.

An excessive distention of the stomach, which in certain cases attains such enormous dimensions that the dilated organ occupies the greater part of the abdominal cavity, and even descends into the pelvis, is so striking a phenomenon at the autopsy that we might naturally expect an early mention of this lesion as soon as the custom of making post-mortem examinations came to be prevalent.

Thus Spigelius (1623) gives an account of a stomach which held thirteen pounds of fluid, and Bonet (1675) of one which extended as low as the symphysis pubis. In 1749 Widmann reported a case in which, besides the dilatation, a constriction of the pylorus was noticed, produced by a cancer situated at that orifice. Since then this mode of origin for the dilatation has been repeatedly observed, and the form of dilatation thus induced, has been distinguished, on account of the clearness of its pathogenesis, from the simple dilatation which is unaccompanied by pyloric stenosis.

The increase in volume affects, at least at first, chiefly the fundus, afterwards the whole stomach. The *mucosa* is sometimes mammillated, and usually thickened, but it may be smooth and thinned, especially at the fundus. Still more striking is the alteration in the thickness of the *muscularis*, which in fact generally composes almost the entire thickness of the wall of the stomach. Occasionally this *thickening* is extremely marked, as in the striking case observed by Mitterbacher a century ago. In nine preparations taken from the pathologico-anatomical collection in this city, Penzoldt found the muscular coat thickened in four instances, in three thickened in some places and normal at other parts, once partially normal, partially thinned, and once everywhere thinned. Skjelderup, in four cases without pyloric stenosis, found the wall only once actually thinned, in two cases half as thick as normal, and once of normal thickness. The microscopical examination usually shows a normal condition of the muscular fibres, but fatty and colloid degeneration have also been observed (Kussmaul and Maier). In some cases the *serosa* has also been noticed to present proliferative changes.

In consequence of the enormous distention of the stomach, the neighboring organs are displaced from their natural position, the intestines backwards, the liver and spleen upwards; moreover, the latter organs, and perhaps also the pancreas, as in Klohss' case, are sometimes atrophied. The spleen especially is very apt to be diminished in size, probably as a result of the pressure exerted by the greatly distended fundus.

When the stomach is much dilated, the beginning of the duodenum is inevitably carried downwards along with the pylorus, although only to a limited extent, and in this way the intestines may slip in between the cardia and pylorus, as was strikingly noticeable in one of my cases (see Fig. 3. p. 312).

This diagram[1] illustrates, moreover, the fact that the part of the greater curvature, which lies nearly opposite the cardia, undergoes the greatest distention, and occupies the lowest position.

In very rare cases circumscribed dilatations of the stomach in the form of diverticula have been observed (Baillie), which have resulted from the continuous pressure of indigestible substances, such as pieces of coin, etc.

Etiology and Pathogenesis.

The most frequent cause of dilatation of the stomach is a stenosis in the pyloric region. As in other hollow organs, when the orifice of exit is constricted, a gradual expansion of the cavity

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[1] Taken from *Penzoldt's* "Dilatation of the Stomach," 1875. *a* represents the cardia, *b* the pylorus; between the two run the greater and lesser curvatures; the former extends at its most inferior part to below the umbilicus; *c* is the enlarged duodenum; *e* the first convolution of the jejunum. The shading around *d* represents the pancreas; *f* shows the abnormal course of the transverse colon.

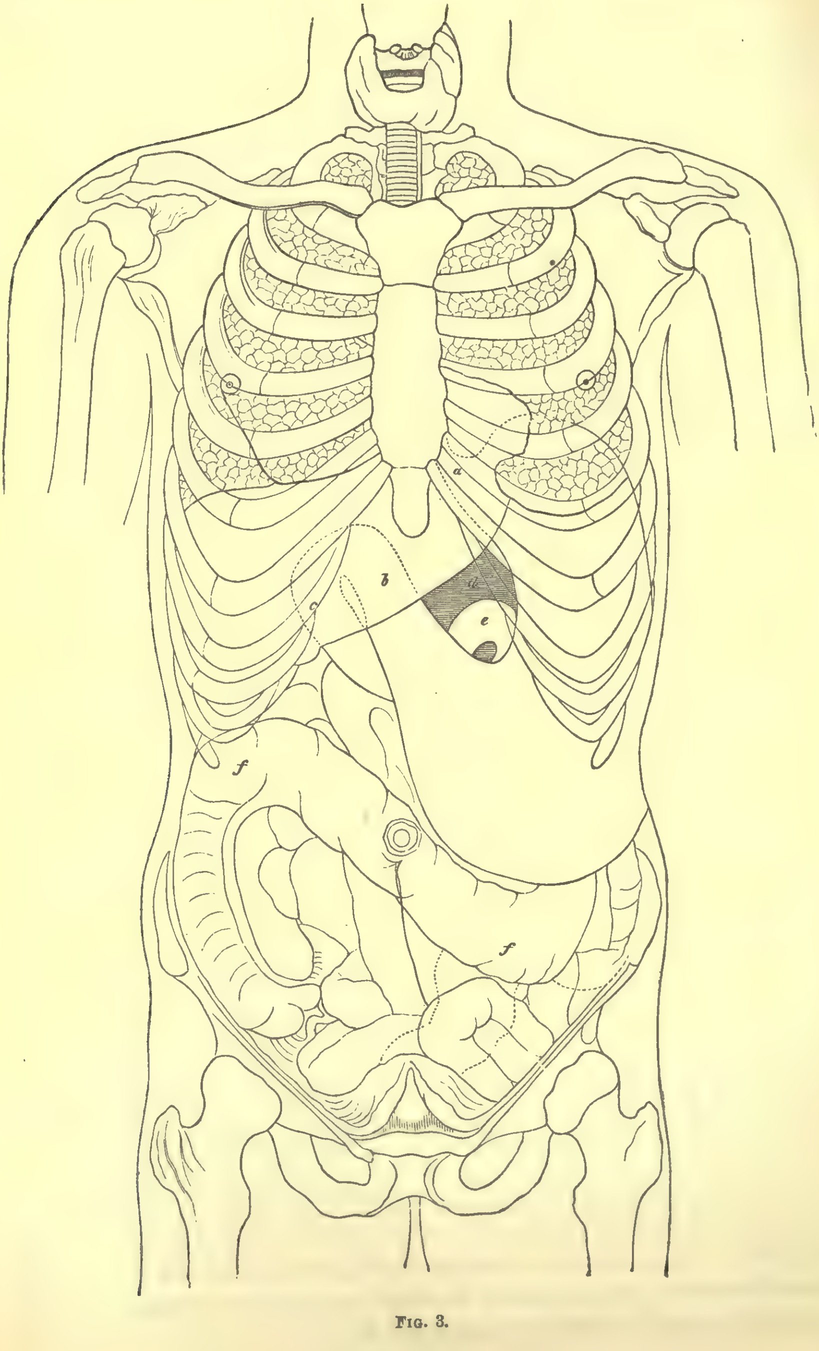

Fig. 3.

is produced by the accumulation of its contents, so also in the stomach the same result takes place when a contraction occurs at the pylorus itself or near it either within the stomach or in the duodenum. The stenosis most commonly results from a pyloric *carcinoma*, much less frequently from the rare proliferations of mucous membrane previously mentioned, or from an hypertrophy of the muscularis limited to the neighborhood of the pylorus. A more common cause is the *ciatrices*, which are left after the healing of a round ulcer at the pylorus or after the healing of ulcerations produced by corrosive ingesta. A narrowing of the lumen of the stomach, especially near the pylorus, may also be occasioned by external pressure, for example, by tumors of the pancreas.

When the stenosis, whatever may be its cause, has continued for a considerable time, an expansion of the cavity of the stomach behind the constriction will inevitably occur, unless this result be prevented by certain compensatory provisions. The simplest of these is *vomiting*, by which the stomach is relieved of its distending ingesta. If the vomiting be incomplete, or too long delayed, or fail to occur at all,[1] the dilatation may be prevented, or at least limited, by the supervention of an hypertrophy of the muscularis sufficient to overcome the obstruction to the ingesta, or by an increased activity of the secretory and absorbent functions of the gastric mucous membrane.

The *hypertrophy of the muscularis* noticed in cases of pyloric stenosis arises in the same way as in the stenosis of other hollow viscera, for instance, the heart, by an adaptation of the size of the muscles to the resistance to be overcome. In many cases the hypertrophy is sufficient to overcome the obstruction, as is evident from the absence of any dilatation of the organ. Thus in a polyphagic patient, in whom Diemerbroeck expected to find an ectasis, the stomach was, on the contrary, remarkably small—half the usual size and of three times its normal thickness. The chief development of the muscles takes place in the neighborhood of the pylorus, while the greater curvature, upon which, as already remarked, the main pressure of the ingesta is exerted, is more or less atrophied.

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[1] This failure at any attempt at vomiting may be explained by a defective irritability of the nerves concerned in the act of emesis. Such a defect may be congenital (the facility with which vomiting takes place differing extremely, as is well known, in different individuals), or it may be acquired in consequence of morbid conditions of the vagus, such as laceration and paralysis, or even complete destruction of the greater part of its fibres. The latter condition could be demonstrated pathologico-anatomically in a case of Traube's, in which almost all the larger branches of the vagus supplying the stomach were found to be destroyed by two large ulcers.

Besides this essentially mechanical mode of compensation, there is still another way in which the results of obstruction may be effectually avoided. The more rapidly and energetically the transformation and *absorption* of the chyme take place, the less food will remain to pass through the pylorus. The transformation of the food will be facilitated by an increased secretion of pepsin, and especially of acids, which, as previously mentioned, favor the formation of peptones. A still greater protection against dilatation will be afforded by an increase of the *absorbent* power of the stomach, because the conversion of the albuminates into absorbable peptones is promoted by an active absorption of the peptones already formed. Such an increased power of absorption will obviously be acquired by the stomach when the muscles have become hypertrophied, for the reason that the process of absorption depends in a large measure upon the muscular movements, and will therefore become more active in proportion to the development of the muscular tissue.

In the failure of these compensatory provisions a permanent dilatation of the organ is the inevitable result. This incompleteness of compensation may be due either to a failure from the start on the part of the muscular hypertrophy and on the part of the increased powers of secretion and absorption to keep pace with the difficulties to be overcome, or else it may arise from the gradual development of a secondary insufficiency of both of these compensatory provisions.

Kussmaul and Maier have shown that in fatal cases of dilatation of the stomach the muscular coat may undergo sometimes a *fatty*, at other times a *colloid degeneration*. On the other hand, the failure of the other mode of compensation was shown in a recent case of my own,[1] by the fact that the fluids withdrawn from the dilated stomach twenty-four hours after their ingestion were apparently in the same condition as when first taken. A deficient supply of gastric juice may probably be inferred also in those cases of dilatation in which obvious benefit is derived from the administration of hydrochloric acid.

The foregoing considerations, which are, in my opinion, of great importance in enabling us to understand the origin of this pathological condition, explain also the occasional occurrence of dilatation of the stomach without stenosis of the pyloric region.

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[1] Volkmann's Sammlung klinischer Vorträge. 3. Serie. 72. Vortrag. 1873.

Dilatation of the stomach will occur whenever a permanent disproportion exists between the quantity of ingesta and the removal of the same. Pyloric stenosis is only the most frequent and most important cause of this disproportion ; the other causes are illustrated in the following cases.

That persons who are in the habit of taking excessive quantities of fluids and indigestible substances into the stomach finally acquire a dilatation of the organ, is a fact of common observation. Thus Hodgkin states that this condition is very common among the vegetarian population of India. The same lesion, to a more or less marked degree, is also a characteristic of gourmands and drunkards. Quite recently I discovered it in a man who had died of diabetes. Generally, however, the difficulty lies not so much in the quantity taken as in the impediment to its removal, arising from weakness of the muscular coat. In this way dilatation may occasionally occur as a result of various acute and chronic affections, such as typhoid fever, tuberculosis, puerperal fever, etc. In chlorosis and anæmia, also, the muscular atony, which is one of the symptoms of these affections, may lead to a gradual dilatation, a mode of origin similar to that described by Sauvages as the cause of meteorism of the stomach. This is probably also the explanation why dilatation is, upon the whole, more common in advanced age than in younger persons.

Dilatation of the stomach may, moreover, be produced by a simple mechanical interference with the complete action of the gastric muscles from adhesions of the wall of the stomach to the liver, intestines, abdominal wall, etc., or from the tension upon the organ by a hernia ; while the dilatation resulting from peritonitis is probably to be explained by the muscular paresis induced by the adjacent inflammation.

When the dilatation develops as a result of anæmia, or of a long-continued gastric catarrh, there is undoubtedly another etiological factor besides the enfeeblement of muscular energy above mentioned,[1] viz., a deficient formation of gastric juice and imperfect absorption of peptones, conditions—the latter particularly—which also result in an excessive accumulation of ingesta in the stomach. In numerous instances of dilatation, however, these two defects of the mucous membrane do not appear to be the original cause; on the contrary, their influence is felt only after the disease has made some progress; then the power of absorption, which is probably at first considerably increased, gradually declines, the peptones remain in the stomach, and the abnormal decomposition of the ingesta with development of gas steadily progresses. Whether the dilatation, observed in connection with diseases of the brain and spinal cord, is due to motor disturbances transmitted through the vagus nerve, must, in the rudimentary state of our knowledge in regard to the relation between the nervous system and the movements of the stomach, remain for the present a matter of doubt. Still more doubtful is the supposed dependence of gastrectasia upon hypochondriasis and hysteria. When these conditions are associated, the dilatation of the stomach is doubtless the primary affection, and the mental disturbance merely the usual result of a protracted gastric disease.

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[1] See also the etiology of gastritis.

Symptomatology and Diagnosis.

Dilatation of the stomach, except when the affection is well developed, presents no symptoms by which it can be recognized with certainty.

The *appearance* of the patient varies according to the duration and severity of the disease. In some instances he is emaciated almost to a skeleton, pale, and hollow-eyed; in others one perceives but little indication of the gravity of the disease—in fact, at first sight the patient gives the impression of obesity, which, however, is entirely due to the bulging of the stomach. Notwithstanding the marked development of the abdomen, the integuments contain but little fat. In proportion to the degree of dilatation, the projection of the abdomen is situated more or less inferiorly, while the epigastric region appears depressed. On closer examination, moreover, the apparent corpulence is in striking contrast with the emaciation of the rest of the body, especially at the nates. Corresponding to this general emaciation, the patient, even early in the disease, experiences a *feeling of reduced physical strength.*

The *appetite*, according to my experience, is diminished in the majority of cases, even when the disease has originated in gluttony. Still, it must always be borne in mind that the appetite frequently remains unimpaired, in spite of the most marked dyspepsia, *indeed that there may even exist an actual craving for food*, a fact which is in striking contrast with the in other respects unmistakably severe derangement of the gastric digestion, and which may assist us in the establishment of the diagnosis.

Besides the loss of appetite, the dyspepsia manifests itself in other symptoms, such as weight and fulness in the abdomen after eating, resulting, perhaps, in part from the dragging produced by the food in the dilated stomach; also nausea, heart-burn, and eructations. These eructations expel either the fluid contents of the stomach, or gases which are sometimes odorless, at other times very offensive, and occasionally even combustible.[1] Finally, many patients suffer from *vomiting*, which is sometimes of a character distinctive of gastrectasia. In quality the vomited matters do not differ in any respect from those met with in chronic gastric catarrh; they are composed of undigested food, sarcinæ, tenacious gray masses of mucus, fatty acids, etc., together with substances of a coffee-ground appearance when the dilatation is caused by pyloric cancer, or a considerable quantity of blood if the case is complicated with gastric ulcer. The *quantity*, however, of the vomited matters may sometimes be an important guide in the diagnosis of dilatation. It is in this affection, more frequently than in any other disease of the stomach, that *enormous quantities of ingesta are discharged at a time* (in one case reported by Blumenthal as much as sixteen pounds!), and not unfrequently such vomiting is an habitual occurrence. It is not, however, an invariable attendant of the disease, and, after it has continued for a long time, it may cease shortly before death. In such cases the cessation of the regularly recurring vomiting is an unfavorable omen, and seems to be due partly to the feeble action of the abdominal muscles, which share in the general exhaustion of the patient, and partly to the increasing paresis of the gastric nerves, produced by their stretching.

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[1] Only three cases of combustible eructations have been reported, one from Friedreich's Clinique (*F. Schultze*, Berliner klin. Wochenschr. 1874. No. 27); the second from Botkins' Clinique (*Popoff*. Ibid. 1870. Nos. 38–40). In both cases the analysis of the combustible gas showed, in addition to N and O—the constituents of atmospheric air—also CO_2 and H; in Schultze's case there were also traces of marsh gas. This gas was comparatively abundant (ten per cent.) in the third case recently published from Frerichs' Clinique (*Ewald*, Dubois-Reichert's Archiv. 1874. S. 217.), and traces of olefiant gas were also found. In this case the eructated gas burned with a yellow illuminating flame, while the flame in the other two cases was colorless.

Dilatation of the stomach is also very generally accompanied by constipation. This symptom depends chiefly upon the imperfect emptying of the stomach. In consequence of the diminution in the quantity of chyme which passes through the pylorus, the bile, and probably also the intestinal digestive juices, are reduced to a minimum, and thus the peristalsis, upon the energy of which the frequency of the evacuations almost exclusively depends, is but feebly excited. The constipation may probably be ascribed in part also to a blunting of the nerves of the overloaded stomach, because a loss of irritability on the part of the gastric nerves is likely to lessen the reflex movements of the intestines; at least it has been observed that when the stomach is emptied at regular intervals by the stomach-pump, the constipation is also spontaneously relieved.

The *reaction of the urine* during this mode of treatment by washing out the stomach may be continuously alkaline, probably because the treatment diminishes the absorption of the gastric acids, and thus withdraws an important factor in the acidification of the urine (Quincke). The *amount* of the urine is variable; in the later stages of the disease it is often diminished—at least this has been the experience of Kussmaul and myself. In my last case of gastrectasia, which I kept for a long time under observation, the urine was very scanty, almost always under thirty-three ounces, and sometimes as little as from thirteen to sixteen ounces. The reduction in the quantity of urine is probably due to the deficient absorption of fluids by the stomach.

This deficiency of water in the blood also gradually induces an abnormal dryness of the muscular and nervous tissues, in connection with which a peculiar nervous symptom has been repeatedly observed by Kussmaul in cases of dilatation of the stomach. The symptom in question consists of *cramps* in the form of tonic contractions of the muscles, alternating with slight clonic convulsive movements. In patients who are suffering from this "drying up of the tissues from loss of water," a single copious vomiting, or the use of the stomach-pump, may excite these cramps by suddenly rendering the dehydration more intense (Kussmaul).

In Kussmaul's cases these painful cramps were confined chiefly to the flexors of the arms, the sural and the abdominal muscles, but sometimes extended also to those of the face and neck. Rolling of the eyes, contraction of the pupils—in fact, even emprosthotonos and disturbances of consciousness were also sometimes observed. The cramps were preceded by painful sensations in the stomach and other parts of the body, together with a sense of oppression and dyspnœa. The *duration* of the attacks is very variable, sometimes continuing for several hours.

In quite rare cases, probably only when the dilatation of the stomach in a downward direction is prevented by the distended intestines or some other obstacles, the expansion takes place upwards to such an extent as to interfere with the movements of the diaphragm, and to excite palpitation of the heart with displacement and increased force of the apex beat (Oppolzer).

The symptoms thus far mentioned, particularly the habitual vomiting of excessive quantities of food, and the maintenance of a good appetite in spite of the presence of digestive disorders, are presumptive, but by no means certain, indications of the existence of dilatation. There are, however, other symptoms which may be regarded as to a certain extent pathognomonic. These lie chiefly in the domain of the *physical examination.*

Inspection discloses a bulging of the abdominal wall, the lower border of which is well defined, and extends downwards as far as the umbilicus, or still further. The appearance of the *tumor* will naturally vary according as the stomach happens to be more or less distended. The tumor will also change in shape when the patient lies upon his side and the contents of the stomach fall to one end of the organ. The boundaries of the tumor may be brought out with special distinctness by the rapid development of carbonic acid gas in the stomach by means of an effervescing powder (Frerichs). When the stomach sinks very low, in consequence of its weight, not only the outline of the greater but also that of the lesser curvature may be seen through the skin, and occasionally also the undulating movements of the organ. These advance from the left towards the pylorus; in one case Bamberger (l. c., p. 296) saw the movements begin with a constriction of the stomach in its middle.

In a case recently under my care, in which the stomach reached as low as the symphysis, I satisfied myself, after weeks of observation, that the movements seen through the thinned abdominal wall, and due apparently to contraction of the stomach, really proceeded from intestinal convolutions which had interposed themselves between the anterior surface of the stomach and the abdominal wall. I would therefore suggest the necessity of caution in the interpretation of this symptom.

On *palpation*, fluctuation, or at least a soft elastic condition of the tumor, can be detected. Moreover, palpation will generally produce a *splashing* sound, audible at some distance, and sounding very much like that produced by shaking to and fro a half-filled bottle. This symptom may of course occasionally occur in the region of the stomach and colon even in the absence of dilatation of the stomach—in fact, it may be artificially produced in healthy persons by making them drink large quantities of fluids rapidly. Still, in my experience, when the sign is *well-marked* and recurs *regularly*, it is always safe to infer the probable existence of dilatation, especially when the splashing noise is readily produced by shaking the patient in an upright position, or when, as I saw very beautifully illustrated in one case, the sound could be excited quite regularly merely by the movement of the diaphragm in deep inspiration. It may also be produced whenever the patient turns quickly upon his side, and not infrequently the symptom becomes a source of constant anxiety and discomfort.

Sometimes the sound is sufficiently loud to be heard at a distance; thus, in one of my cases, the nurse who slept in the same room with the patient was unable to sleep on account of the loud splashing noise produced whenever the patient turned from one side to the other.

The sounds heard on applying the ear to the wall of the abdomen, while the fluid drank by the patient falls to the bottom of the cavity of the stomach, are of but little value for an exact diagnosis, and the same is true of the feeling described by the patient, that, on drinking, the fluid falls "low down in the abdomen."

Much more significant than the signs above described are those obtained by *percussion.* The percussion note varies according to the contents of the stomach, being sometimes dull, sometimes tympanitic, and occasionally presenting even a very marked metallic tone. This tone has a comparatively deep quality, on account of the size of the air-containing space percussed, and, under some circumstances, may extend considerably beyond the usual limits of the stomach note, downwards to below the navel, and upwards towards the axilla. The lower portion of the tumor, which corresponds to the greater curvature, and the full outlines of which are often easily distinguishable through the abdominal wall, sounds dull on percussion when the patient is upright, because a dilated stomach probably always contains at least a small quantity of fluid.

When the patient is placed upon his back, the dull region lying to the left of the umbilicus, and corresponding to the inferior portion of the cavity of the stomach, disappears entirely, and is replaced by a tympanitic sound, because the solid and fluid contents sink to the posterior wall of the organ. This behavior of the percussion note shows, at all events, that we have to deal with a large air-containing organ, which, besides air, contains also a certain amount of fluid, changing in position with the posture of the patient; but, whether this hollow organ is the stomach, or a portion of the intestinal tract, particularly the transverse colon, percussion alone does not enable us to determine. The question, however, may be decided by a simple manœuvre. If a healthy man, while fasting, be made to drink water (a quart), a strip of dulness makes its appearance *above* the umbilicus (see Penzoldt's experiments, l. c., p. 48); if the strip of dulness appear below this point, dilatation of the stomach very probably exists, and the diagnosis becomes more certain if the stomach-tube be used, and fluid be introduced and withdrawn at discretion. If, with this manipulation, a dull strip regularly form below the umbilicus, and disappear every time the fluid is withdrawn, dilatation of the stomach may be diagnosticated with a considerable degree of certainty. *Finally, all doubt is removed if the stomach-tube can be felt below the horizontal line drawn from the anterior superior spinous process of the ilium on one side to that on the other.*

The first occasion on which I felt the point of the tube with surprising distinctness, near the symphysis, was in a patient the history of whose case is briefly given below, because it affords a very marked illustration of dilatation of the stomach, and contains also an account of some further experiments in regard to the palpability of the point of the instrument.

Mrs. K., forty years of age, wife of a worker in metals, admitted January 19, 1875, states that four years ago, while enjoying perfect health, she vomited blood; her present illness, however, began in the last week of April, 1874. Her symptoms consist of vomiting, three or four hours after eating, headache, acid eructations, alternate chilliness and sweating, a sense of fulness, and pains in the epigastrium or over the whole abdomen, especially after taking a hearty meal. Vomiting always relieves these pains, while change of position entirely fails to modify them. The matters vomited contain nothing abnormal. Patient states that, sometimes, on stooping after she has taken food, "water runs into her mouth." The appetite has always been good, notwithstanding these digestive derangements, but she has been afraid to yield to it. The stools have always been solid, and have frequently been retained for weeks at a time. On defecation, a sensation is felt, as if the escape of the fæces were obstructed. She is unaware of any cause for her disease; has never committed excesses in eating; prefers her food hot.

Present state: Abdomen pendulous, and distinctly more protuberant on the left than on the right side. Over this prominent region there are very striking vermicular movements, which at first sight give the impression of contractions of the stomach, but, on more careful examination, must be ascribed to the peristalsis of intestinal convolutions, which can be seen every now and then to interpose themselves between the stomach and the anterior wall of the abdomen.

Palpation gives unmistakable fluctuation, and a succussion sound which can be heard at considerable distance.

Percussion, with the patient in the upright position, reveals dulness in the left half of the abdomen, beginning at the umbilicus and extending downwards halfway to the symphysis. This dulness disappears when she lies upon her back, and extends higher up when fluids are taken into the stomach.

On *auscultation* a humming noise can be heard, which may best be compared to the crepitant sound produced by carbonic acid after uncorking a soda-water bottle (fermentation murmur),[1] and which becomes much more distinct after an effervescing powder has been taken.

After injecting two thousand c. ctm. of water by means of the stomach-pump, two thousand eight hundred and fifty c. ctm. of fluid were withdrawn, containing large quantities of sarcinæ, and some whortleberries which the patient had eaten about a week previously.

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[1] Pauli, Oppolzer, Penzoldt, l. c.

The lower limits of the *lungs* in the papillary line stood at the upper borders of the seventh ribs. *Apex of heart* beat in the fifth intercostal space, with a heaving motion, which could also be clearly felt in the sixth space; some extension of cardiac dulness to the right.

Urinary secretion scanty—almost always below one thousand c. ctm. daily.

The *weight of the body* remained quite uniform during her six weeks' stay in the hospital.

After a stomach-tube, about seventy ctm. (twenty-six inches) long, had been introduced its full length without resistance, I could feel its point with surprising distinctness through the abdominal wall to the left of the symphysis, and could easily push the point against the umbilicus.

Whenever this examination was repeated, the same result was obtained, even up to the time when she left the hospital, at the end of February—not cured, but, at least relieved of the vomiting and other distressing dyspeptic symptoms which had brought her to the hospital. *The point of the sound could still be felt just above the symphysis, and on one occasion (the middle of February) was even caught between the finger introduced into the rectum and the hand compressing the abdominal wall!*

Further experiments upon six apparently healthy individuals showed that in two the end of the tube could be distinctly felt considerably below the umbilicus, and in the other four near this point, or a finger's breadth below it. It is evident, therefore, that in a normal condition of the stomach the sound can be introduced at least as low as the umbilicus. In order to decide whether the introduction of the instrument in two of the cases as low as three fingers' breadths below the umbilicus was due to a great distensibility of the lower wall of the stomach or to a latent dilatation, another series of experiments were made upon the cadaver.

These experiments demonstrated that the natural position of the greater curvature is not the lowest point to which the instrument can be introduced; on the contrary, *that in the cadaver the lower wall of the stomach opposite the cardia can be tilted downwards a considerable distance, and can even be forced nearly as low as the horizontal line between the anterior superior spinous processes of the ilia.* When this force was employed, the entire lower wall of the stomach was drawn down towards this most dependent portion of the greater curvature, in which the end of the sound was engaged, in such a way as to give the appearance of the finger of a glove.

In the living subject a distention of the stomach to such an extent as this is under normal circumstances incredible, because even in the cadaver this low introduction of the tube was sometimes accomplished only by injuring the mucous membrane, and, in fact, even perforating the wall of the stomach which presented no signs of cadaveric softening. It is safe, therefore, to infer that the lower wall of the stomach can ordinarily be extended by the tube only as far as the umbilical horizontal line, and that a further extension is practicable only when the gastric mucous membrane is quite insensitive. The nearer the point of the instrument can be felt to the horizontal line between the ilia, the more certain is the *diagnosis of gastrectasia, and when the point is felt below this level the certainty is absolute.*

That the tubes usually employed, even the longest of them (about seventy ctm.), must in such cases be introduced their entire length, is shown by a simple estimation of the distance from the incisor teeth to the symphysis; and the mere fact that the tube passes its full length without meeting any obstruction, is *primâ facie* evidence that we have to deal with a dilatation of the stomach.[1]

The more important symptoms and means of diagnosis of dilatation of the stomach may be recapitulated as follows:

Habitual vomiting of the contents of the stomach, frequently in very copious quantities, with great relief to the patient; the regular occurrence of a loud splashing succussion sound on quick movements of the trunk; the increased extent of the deep tympanitic note both before and after artificial distention of the stomach; the location below the navel of the dulness due to the fluids in the stomach, the line of dulness moreover following the changes of position assumed by the patient; the controlling of the results of percussion by the introduction of fluid into the stomach and the withdrawal of the same by means of the stomach-pump; the length to which the tube can be introduced; and, finally, the tangibility of the point of the instrument through the abdominal wall near the symphysis.

From this summary we may conclude that, in most cases, at least when the dilatation is considerable, there can be no difficulty in making a certain diagnosis—in fact, I think I am warranted in expressing the hope that with the aid of these expedients, particularly the palpation of the point of the stomach-tube, we shall be able to recognize the existence of more or less *latent* conditions of dilatation, which have hitherto escaped our powers of diagnosis. The following case will serve as an illustration.

In a patient, who had apparently no affection of the stomach, the end of the stomach-tube had been felt *below* the umbilical horizontal line, and the suspicion had been unavoidably entertained that we had to deal with a latent dilatation of the stomach. He was suffering from diabetes insipidus, without any symptoms of gastrectasia, such as eructations, pain in the stomach, etc. After having been discharged on the nineteenth of February, he returned on the twenty-seventh inst., complaining of dyspepsia. During the eight days he was out of the hospital he had been taken with vomiting, and at one time he had thrown up three pints of fluid tasting like bile. Another examination with the tube was immediately made, and it was discovered that now the end of the instrument could be felt decidedly further down than previously—that is, *below* the horizontal line between the ilea (eight ctm. below the umbilicus instead of between three and four, as on the former occasion). There was now no longer any doubt in regard to the existence of gastrectasia, especially as the lower level of the fluid in the stomach could be recognized by percussion as lying three ctm. below the umbilicus. After pumping out the contents of the stomach, this dulness below the umbilical horizontal line at once entirely disappeared. *The originally latent dilatation of the stomach had, therefore, progressed during this interval sufficiently to become recognizable.*

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[1] According to Penzoldt's measurements, the instrument can be introduced in healthy persons about sixty ctm.; in cases of dilatation about seventy ctm. (which corresponds to the length of the spinal column from the occiput to the coccyx).

Course and Prognosis.

Dilatation of the stomach runs a course of more or less protracted duration, according to the nature of the cause which has produced it. Thus, when it is conditioned by a carcinoma pylori, its duration coincides with that of the cancerous disease; if the stenosis be of a non-malignant character, the dilatation may last for years; while if it be developed, as a result of a cachectic condition, such as chlorosis, an improvement may reasonably be expected when the fundamental affection is removed.

The prognosis is, upon the whole, *unfavorable*, although, thanks to the new method of treatment by the stomach-pump, the chances of relief have become incomparably better than formerly. My own experience, however, is decidedly opposed to the too optimistic expectations which were at first entertained from the therapeutic use of the stomach-pump. At all events, the cases in which the stomach resumes its normal size, and the pyloric tumor can be felt to move upwards, as Kussmaul has several times observed, are rare, and I am convinced that such reported instances of cure in marked gastrectasia will become still less frequent when it becomes more customary to compare the position of the introduced tube at the time of apparent recovery, with its position at the beginning of the treatment. The history of the case of the female patient previously given shows most conclusively how cautious one ought to be in judging of the results of treatment in dilatation of the stomach;

under the use of the stomach-pump for four weeks the distressing symptoms of dyspepsia were strikingly relieved, and the general health satisfactorily restored; and yet, when the patient, at her own request, left the hospital, the point at which the end of the tube was felt was not a single centimetre higher than at the beginning of the treatment.

The longer the dilatation has lasted, and the greater its dimensions, the longer, also, the stomach-tube is used without restoring the expulsive power of the stomach or the regular action of the bowels, the more unfavorable must be the prognosis, and the more inevitable the conclusion, either that the pyloric obstruction is irremediable, or that the muscular paresis of the stomach is anatomically due to a degeneration of the cells of the muscles. In such cases the condition of the patient becomes more and more pitiable, all food disagrees with him, the power of vomiting is lost, dropsy occurs, and death finally occurs from marasmus. In rare instances laceration of the tensely distended stomach may occur, and then death ensues from perforation-peritonitis (Duplay).

Obviously, also, the prognosis is always more serious when the dilatation is accompanied by, or is due to, other constitutional or gastric diseases, such as tuberculosis, chlorosis, cancer, ulcer, or chronic catarrh of the stomach, etc. (See Etiology.)

Treatment.

In this affection therapeutic measures suggested by the *causal indication* are usually of subordinate importance. Since, however, the development of dilatation of the stomach is favored not only by mechanical causes, but also to a marked degree by constitutional conditions of a debilitating character, a *roborant* diet, iron, etc., are occasionally an important part of the treatment. At the same time it is to be borne in mind that polyphagia is in certain cases the essential cause of the disease, and that in all cases the stomach is taxed beyond its powers of digestion.

It is in general advisable, therefore, that the patient should take but small quantities of easily digested food at a time, and that the use of fluids should be restricted as much as possible.

It is interesting to notice that van Swieten (Comm. ad Aphorism. § 605, 10) lays down regulations of the same purport: "Parca tantum copia cibus et potus assumatur, ut omnis distentio ventriculi caveatur: sic enim sensim fibræ ventriculi nimis distractæ pristinum robur acquirent."

Canstatt (l. c. III. 288) long since recommended "the frequent withdrawal of the accumulated fluid in the stomach by means of the stomach-pump" in cases in which the patients had lost the power of vomiting. But the systematic use of the stomach-pump for this purpose, in cases of marked gastrectasia, was first suggested recently by Kussmaul, who was led to adopt the practice by the *à priori* consideration that it must be possible "by means of the pump to empty the stomach completely, and probably to restore its capacity to contract to its normal dimensions." The results of this treatment exceeded all expectations, and attracted universal attention and approbation; indeed, it may be said that with the introduction of this practice, the treatment of dilatation of the stomach has, for the first time, become a rational one, and that all other remedies in use sink by comparison into the second or third rank. The particular mode by which this evacuation is to be effected, whether by the stomach-pump, as recommended by Kussmaul, or by a syphon apparatus (Ploss, Juergensen, L. Rosenthal, and others) connected with the stomach-tube, is, in my judgment, of quite subordinate importance. My own experience is, however, decidedly in favor of the stomach-pump.

Until two years ago I used and recommended the syphon-tube almost exclusively, because I was convinced by my own experience that the gastric mucous membrane might be aspirated in the fenestra of the tube by the suction force of the pump, and thus lacerated. (See Ziemssen's case, Deutsches Archiv. X. 65.) Still, very few instances of this unfortunate accident have been reported, notwithstanding the increasingly frequent use of the instrument.

On the other hand, the syphon has unquestionably the disadvantage that it can withdraw from the stomach only fluids, or at least only substances which are very nearly fluid, so that one is obliged finally to resort to the pump to remove the remnants of the ingesta. But in the treatment of gastrectasia, the removal of the ingesta to the fullest extent possible is altogether the most important indication, and therefore that instrument is to be preferred by which this result can be most certainly attained. If it be urged in favor of the syphon apparatus that, unlike the stomach-pump, it can be used by the patient himself without danger, I must dissent most decidedly from this opinion. The operation of washing out the stomach, when intrusted to the patient alone, without the supervision of the physician, is attended with so much risk from the unavoidably awkward movements of the patient, in connection with the protrusion of the lower wall of the stomach by the end of the tube, as above described, that I cannot but regard the relegation of the operation to the patient himself as extremely hazardous.

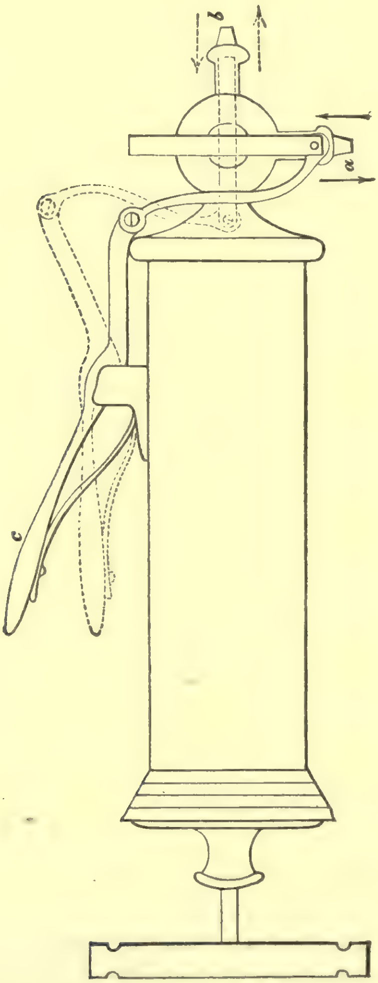

Fig. 4.

I would recommend, therefore, that the pump be used for ordinary purposes, and the syphon only in those cases in which the contents of the stomach consist entirely of fluid, or in which a complete evacuation is unnecessary—for instance, when the tube is employed only for diagnostic purposes.

The best instrument for removing the contents of the stomach is the pump [to be obtained of any instrument-maker in England or America, where it has long been in use as the ordinary valve stomach-pump.—Trans.] described on page 328. It is supplied with a lever, like a trumpet-valve, which is depressed by the thumb of the left hand holding the instrument. The movements of this lever effect a quarter turn of the stop-cock towards the entrance and exit tubes respectively.

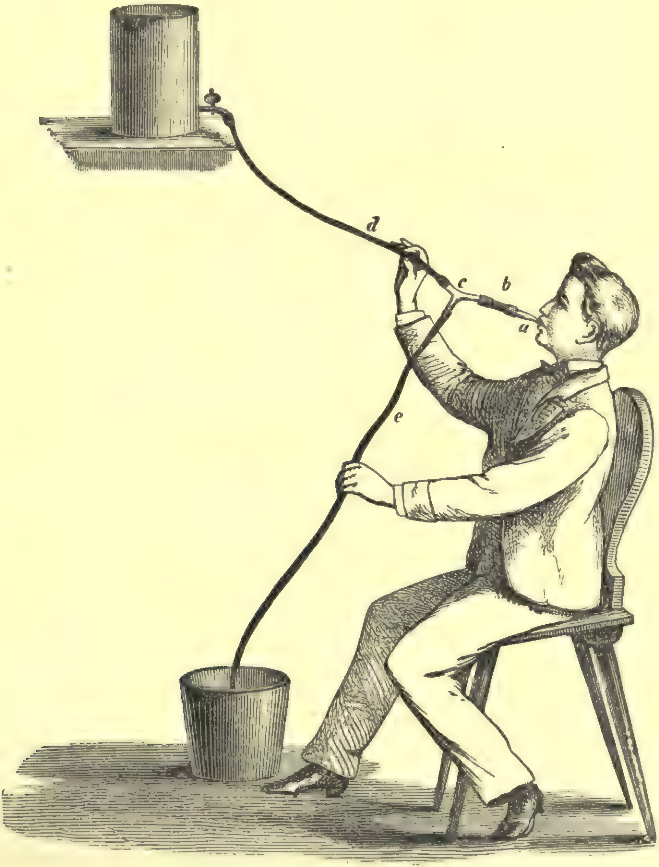

Fig. 5.

After the rubber sheath of the tube *a* is fitted over the end of the stomach-tube, the piston is drawn out; the contents of the stomach now pass directly into the pump; the lever *c* is then depressed, and the position of the stop-cock is thereby changed, so that the interior of the pump is shut off from the tube *a*, and communicates with the escape-tube *b*.

The simplest *syphon apparatus* consists of a funnel and a long tube attached to the end of the stomach-tube. The funnel is first elevated and water poured through it into the stomach; on lowering the apparatus, the gastric fluids escape. Somewhat more complicated, but still sufficiently simple, is the following contrivance, which agrees in principle with the one recommended by L. Rosenthal, and which I use exclusively.

Over the upper end of the stomach-tube *a* is fitted a short piece of rubber tubing, which is connected with one limb of a glass triangle, the two other limbs of the latter being attached to long rubber tubes. One of these passes to the stop-cock of an irrigator, the other into a receiving vessel. The physician (or the patient himself) first allows the water to flow down into the stomach through *d*, the tube *e* at the same time being compressed. When the column of water has reached the stomach, the pressure on the tube *e* is relaxed. The water from the irrigator now rushes into the receiving vessel, and carries with it also the fluid out of the stomach through the stomach-tube. If the stop-cock be now turned, or the tube *d* be compressed, the contents of the stomach flow out until the stomach is emptied, or until a stoppage in the stomach-tube occurs. In the latter case, if *e* be compressed, and *d* again relaxed, as at the outset of the operation, the obstruction, if not too great, will be washed away, and the outflow restored.

The stomach-tube ordinarily used for washing out the stomach I regard as unsuitable for this purpose. Not only is it too short to reach the greater curvature of a dilated stomach, and the fenestra too narrow, but the funnel-shaped end is unnecessary, because it only increases the difficulty of stretching the rubber tubing of the stomach-pump over it; while, for the purpose of administering food, the attachment of a simple glass funnel would answer as well.

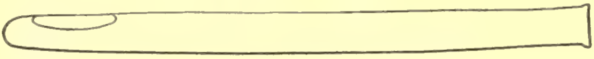

FIG. 6.

The advantages of the treatment of gastrectasia by the stomach-pump are obvious.

In the first place, the systematic evacuation of the ingesta, which often accumulate in enormous quantities, *prevents any further taxing* of the wall of the stomach, and restricts the injurious influence which they exert upon the structure and functional powers of the muscularis, while, under favorable circumstances, if the contractile power be not entirely lost, the stomach may even contract again to its normal dimensions.

As the contents of the dilated stomach usually consist of a fermenting and extremely acid fluid, *the withdrawal of the same relieves the mucous membrane of a permanent source of irritation, and, moreover, prevents the food taken from being constantly decomposed, as heretofore, by the fermenting substances retained in the stomach.* Under this treatment, therefore, the dyspeptic symptoms are found to disappear very rapidly.

With the restoration of the contractile power of the muscular coat and the removal of the compression from the lymphatics and blood-vessels, the stomach again becomes able *to absorb completely the products of digestion.* The peptones, therefore, no longer remain in the stomach to interfere with the further digestion of the food, and the nutrition of the patient steadily improves.

Under this treatment, moreover, the *vomiting* and *obstinate constipation* are relieved, the latter probably because, on the one hand, the muscularis soon becomes able to drive the ingesta through the pylorus, and because, on the other hand, the re-established irritability of the gastric nerves enables the latter to excite a more vigorous peristalsis of the intestines.

Finally, there is a very striking *improvement in the subjective condition* of the patient, since the nausea, heart-burn, vomiting, and sense of fulness usually disappear very rapidly, and are replaced by a feeling of great relief.

While the experience of the past five years with the treatment of gastrectasia by pumping out the stomach has been upon the whole very gratifying, it must be confessed that there are *limitations to the effectiveness of the treatment.* This fact was not overlooked by Kussmaul; in his very first recommendation of the stomach-pump he reminds us that in cancerous or marked cicatricial stenosis of the pylorus, or in cases attended by an irreparable degeneration of the wall of the stomach, a certain amount of relief may be expected from this treatment, but at all events not a cure.

Moreover, cases sometimes occur in which the use of the stomach-pump is either unjustifiable or entirely impracticable, *e. g.*, when, in addition to the stenosing cicatrix at the pylorus, there is also good reason to infer the presence of recent ulcers, or when the patient exhibits an insuperable repugnance to the introduction of the tube, or, finally, when the mere attempt at introduction excites intense alarm and spasm of the muscles of deglutition. In such cases we are obliged to resort to other means by which the overloaded stomach may be completely or at least partially emptied.

The most natural substitutes for the pump would be *emetics.* But aside from the discomfort to the patient from their administration, it is probable that they never secure a complete expulsion of the fermenting ingesta. Moreover, when the patient is much reduced by the disease, and the muscles have lost their tonicity, it may be very difficult or even impossible to excite vomiting. We are generally obliged, therefore, when the stomach-pump cannot be employed, to resort to *cathartics*, which carry off the ingesta through the pylorus. Both Köhler and Kussmaul have obtained surprising results in some cases from the use of drastic pills (extract of colocynth, scammony, etc.), while I myself am in the habit of using Carlsbad water for this purpose in the milder as well as in the severe cases. This water, as has recently been shown with some degree of probability by the experiments of Penzoldt[1] in a case of dilatation of the stomach, not only expels the contents of the stomach into the intestines, but also tends to relieve the complicating gastric catarrh. If anæmia be present, and if the dilatation seem to be due to this cause, or at least to be kept up by it, I prescribe hydrochloric acid (to be taken a few hours after eating), partly for the purpose of assisting the transformation of the food in the stomach, and partly for the purpose of promoting the expulsion of the fluids in the stomach by increasing their acidity.

But whatever the method by which the systematic evacuation of the stomach is accomplished, permanent benefit cannot be expected without a careful regulation of the *diet.* Especially must the *quantity* of the food and drink be limited, while in general the diet must be of a dry and "easily digestible" character, so as to avoid, as far as possible, any distention of the stomach. For this purpose, also, the patient should wear an abdominal bandage (J. Frank, Kussmaul), and should lie upon his right side during the greater part of the day, in order to facilitate the escape of the food from the dilated stomach.[1]

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[1] l. c., p. 76.

To restore the tonicity of the over-worked gastric muscles, the *constant current* may be applied to the epigastrium. I have obtained results from this mode of treatment, which at least call for further trials of its value. Nux vomica has also been repeatedly recommended with the same view (Duplay).

For the purpose of promoting the secretion of the gastric juice and of thus increasing the powers of digestion, the different bitters may be used, or trial may be made of cracked ice, either alone or in combination with milk, as in Pétrequin's ice-milk treatment.

When the above-described plan of treatment is carried out, special prescriptions for the relief of individual symptoms will seldom be required. If, however, the vomiting, acid eructations, or other symptoms be particularly troublesome, the general course of treatment may be supplemented by the remedies which have been fully considered in the chapter on Gastritis.

Contraction of the Stomach. Angustatio Ventriculi.

As the most frequent *cause* of dilatation of the stomach is a stricture of the pylorus, so also a stricture of the cardia or œsophagus is not unfrequently the occasion of a *contraction* of the organ, since the stomach, like other organs of the body, especially the hollow ones, gradually adapts itself in size to the bulk of its contents and the amount of work it has to perform. Contraction of the stomach is found also in cases of long fasting, or when food has been taken in insufficient quantities. The latter cause is met with more particularly in women. Frequent child-bearing and similar exhausting conditions occasionally produce in the weaker sex a premature marasmus, one of the important results of which is a contraction of the stomach similar to that which occurs in senile marasmus. Of this latter condition the contraction of the stomach is so common an expression that many physicians, as Schönlein[1] and Canstatt,[2] regard true "marasmus senilis" as essentially an atrophy of the stomach and intestines.

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[1] Penzoldt, l. c., p. 77.

The cavity of the stomach may, of course, be narrowed also by tumors in the vicinity or implanted in the wall of the organ at some other part than the pylorus; here their effect would be to produce dilatation. Contraction may also result from the shrinking of the extensive cicatrices left after the healing of ulcers.

The cavity of the stomach is sometimes so much reduced in size as to scarcely hold a hen's egg,[3] or it may even appear narrower than the duodenum (Haller's case).[4] The wall itself may be thinner or thicker than normal, generally the latter; a thinning of the wall usually results in an atonic dilatation of the organ.

The *clinical picture* presented by the disease under consideration is by no means characteristic, for all the symptoms which are regarded as in some degree distinctive—the distress on taking large quantities of food, the vomiting, the constipation, and the emaciation—are obviously too ambiguous to allow any conclusion to be drawn from them. So also but little importance can be attached to a diminution in the extent of the resonance on percussion, or to a sinking in the epigastrium. A diagnosis is possible only with the aid of the stomach-tube, which, in a case of contraction, will strike the greater curvature sooner than is normal,[5] and even when strongly pushed downwards, will not be felt lower than the border of the ribs. Moreover, if the stomach be first emptied by the pump, and then water be poured into the cavity, the fluid soon regurgitates through the tube, while the lower limit of the dulness produced by the fluid is always situated above the border of the ribs.

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[1] Allg. u. spec. Pathologie. St. Gallen, 1841. I. S. 89 u. 91.

[2] Die Krankheiten des höheren Alters, etc. 1839. II. S. 276.

[3] S. Löseke. Other remarkable instances of contraction of the stomach are recorded chiefly in the older works. See particularly Jos. Frank, Præcepta pars. III. Vol. I. Sec. 2. p. 222. 1835.

[4] Elem. Physiol. Lib. XIX. Sec. 1. § 3.

[5] This striking of the lower end of the tube against the wall of the stomach can sometimes be felt by the physician himself; at other times the patient will be able to state the moment of striking with the greatest positiveness.

The *prognosis* varies according to the cause of the disease, being of course most serious when the contraction is produced by cancer, and most favorable when it results from insufficient nourishment.

The *treatment*, so far as any is required, consists only in adapting the diet to the contracted condition of the stomach, and in building up the general health of the patient by tonics.

Anomalies in the Shape of the Stomach.

These alterations in shape are some of them congenital and others acquired after birth. Among the *congenital* malformations we find, besides the above described abnormal diminution in size, *constrictions of the stomach* by which the cavity is separated into two or more sacculated partitions, and various degrees of *atresia of the pylorus.* In the latter condition the stomach is sometimes connected with the duodenum only by a cord, or is entirely separated from it.

The *acquired* malformations are usually produced by tumors situated in the wall of the stomach, and especially by the contraction of extensive cicatrices.

These alterations in form, except so far as they produce results which have been previously discussed, possess no clinical, but only a pathologico-anatomical interest. The same remark will apply also equally to the anomalies in position mentioned below.

Anomalies in the Position of the Stomach.

These also are partly congenital, partly acquired. Of the congenital malpositions the most important are: a displacement of the stomach into the thoracic cavity, in consequence of a complete or partial absence of the diaphragm,[1] and a complete reversion in the position of the organ and its individual parts in connection with a general transposition of the viscera. In the latter case the diagnosis presents no difficulty, if the abdomen and chest be carefully examined, while the displacement of the organ into the left thoracic cavity can be confounded only with pneumothorax, from which it can best be distinguished by noticing the increase and decrease of the limits of dulness according as fluid is introduced or withdrawn through the stomach-tube.

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[1] A collection of cases of displacement of the stomach with diaphragmatic hernia is to be found in Schmidt's Jahrbücher. 1856. Bd. 89. S. 169. Another case of extreme interest is described by Rokitansky (Lehrb. III. S. 181).

Among the *acquired* malpositions may be mentioned : dislocations of the stomach by large tumors in its vicinity and by a distended transverse colon, the inclusion of the stomach in large umbilical and scrotal hernias, and its situation in the thoracic cavity in cases of rupture of the diaphragm. Distortions of various kinds may also be produced by adhesions to neighboring movable organs. Finally, the outlines of the stomach are altered in position by the prolapse of the organ during the development of a carcinoma or a gastrectasia.

Such changes of position can be best recognized by the use of the stomach-tube, by means of which alternate dulness and resonance can be produced at will in the region abnormally occupied by the stomach, through the introduction and withdrawal of fluids. Finally, the palpation of the end of the tube, in an entirely abnormal situation, will remove all doubt of the existence of a malposition of the stomach.

Whenever the stomach is displaced, derangements of digestion are probably never entirely absent; sometimes, however, they are unimportant, and then the pathological position of the organ may manifest itself in other ways, *e. g.*, by dyspnœa after hearty meals or on lying upon one side.

Engel (Wiener medic. Wochenschrift. 1857. No. 41. S. 738) has found an actual *intussusception of the stomach* in cases in which only the left half of the stomach was distended by gas, the pressure exerted by the colon being only moderate. Under such circumstances "the distended left half of the stomach is pushed over the strongly contracted right portion, and the pylorus appears to be *invaginated* into the cardiac half of the organ."

Solutions of Continuity.

Literature of ruptures of the stomach: For the older literature see *Plouquet's* lit. med. dig. "Ventriculi ruptura."—*Lefèvre*, Recherches médicales pour servir, etc.—perforations spontanées. Archives générales de Méd. 1842. Août et Septbr. S. 377. An exhaustive work with experiments and a historical development of the theory of ruptures of the stomach; containing also a full bibliography and a compilation of cases.—*Romberg*, Ueber die Tödtlichkeit der Magenwunden, etc. Originalabhandl. in Schmidt's Jahrb. 1845. Bd. 46. S. 231 spec. 237.—*A. Poland*, Contusions of the abdomen. Guy's Hosp. Rep. 3. Ser. Vol. IV. p. 123. Refer. in Schmidt's Jahrb. 105. S. 74. 1860, with a compilation of several cases.—*L. Casper*, Praktisches Handb. der gerichtl. Medicin. II. Band. 1864. S. 276.—*Newman*, Case of general emphysema, etc. Lancet. Dec. 1868. p. 728.—*Buist, J. Somers*, Rupture of the stomach and the spleen from a fall without external evidence of injury. Americ. Journ. of Med. Scienc. 1870. Canstatt's Jahresb. II. S. 157. The literature is not included which treats of cases in which the rupture resulted from gastric disease with erosion of the wall of the stomach.

Perforation of the stomach, as is well known, is a not infrequent termination of various gastric diseases accompanied by erosion of the coats of the organ, such as cancer, ulcer, and, in rare cases, softening and dilatation of the stomach. The finally resulting rupture in these cases is merely one of the symptoms of the antecedent disease, and, as such, has already been considered in the respective chapters. Another form of rupture of the stomach, however, still requires special mention, viz., that which occurs in an apparently healthy stomach, and which may be termed *gastrorhexis, or rupture of the stomach in the strict sense.*

The question may be raised, whether, in view of the great elasticity of the wall of the stomach, a laceration of the perfectly healthy organ is ever possible during life.

This doubt is to a certain extent justified by the results of experiments upon the cadaver, since it is very easy to satisfy oneself that water may be poured into the stomach so as to distend it *enormously* without producing any noticeable solution of continuity. But if this excessive distention be carried beyond a certain limit, the serous coat upon the surface of the tense organ is seen to give way, and fissures corresponding to those of the serosa take place in the mucous membrane, while the muscular coat remains apparently intact. If the distention be effected by means of a bellows, an emphysema of the wall of the stomach is produced, and finally complete rupture, the laceration taking place most readily at the fundus and in the vicinity of the cardia along the lesser curvature (Lefèvre, l. c., p. 412). The apertures thus made are round, with fimbriated edges, or at other times look as if the part had been cut out with a punch—at least these were the results of Lefèvre's experiments.

The literature of this subject shows unquestionably that rupture may occur in a perfectly healthy stomach ; still this accident must certainly be regarded as among the rarest of events, and in future cases it will be necessary to show by microscopical examination that there has been no fatty degeneration of the muscular coat. Care must be taken also to estimate what share in the perforation is due to the excessive distention of the wall of the stomach, and what to the ante-mortem gastromalacia. The decision of this question turns upon the presence of rents in the serous and mucous membranes near the perforation.

External violence to the abdomen, in the form of blows, kicks, falls, being run over, etc., usually produces a rupture of the spleen, or more frequently of the liver ; but the cases are not very rare in which the laceration under such circumstances affects the stomach alone or in connection with other organs.

Furthermore, cases have undoubtedly been observed in which the stomach has ruptured after a hearty meal or after eating indigestible food. Cases of this kind recorded by the older writers, before Cruveilhier's recognition of gastric ulcer, are of course open to suspicion.

Among the predisposing causes of rupture of the stomach we must evidently include also the difficulty with which certain individuals vomit. When the food fails to escape through the pylorus with the proper rapidity, and gases are formed from the decomposition of the ingesta, the distention of the stomach will become more and more excessive, until finally rupture will occur, unless it be prevented by vomiting.[1]

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[1] Lefèvre mentions as analogous the rupture of the stomach which is met with in the horse, an animal, which, as is well known, vomits with great difficulty. Mesnard's case (ibid. Obs. IV.) is an excellent illustration of the importance of free vomiting at the proper moment, in order to prevent the occurrence of rupture of the stomach. The patient felt as if the fluid drank passed down only as far as the middle of the chest and was then immediately regurgitated. But more probably the opening of the cardia in the movement of vomiting lasted but for a moment, and was followed by a "spasmodic con-

The train of symptoms which has been observed in cases of fatal rupture of the stomach is briefly as follows: a few hours after eating, usually during the night, the patient is attacked with a severe pain and weight in the epigastrium, which becomes much distended. Nausea and ineffectual attempts at vomiting ensue. A dreadful terror overpowers him, he tosses to and fro, bends himself together, and can obtain no relief by change of position. Now and then the torture is relieved by intervals of comparative ease, but only to return with renewed violence. The pulse is regular, slow, and hard. All at once the scene becomes altered; the patient is himself sometimes conscious that a change has taken place in the abdomen; the hitherto tearing pain acquires more of a burning character and shifts in position, the abdomen swells and becomes exquisitely tender, the pulse frequent and small, the skin cold and covered with a clammy sweat, and the face distorted—the picture of perforation-peritonitis becomes more and more complete. If the parietal peritoneum have also been torn in consequence of the excessive tension, a general emphysema of the skin may ensue (Newman). Death finally results, the patient retaining full consciousness up to the last moment.

The *prognosis*, after perforation has taken place, is of course absolutely fatal, and the *treatment* merely that employed for perforation-peritonitis in general. In the first stage, however—that preceding the laceration—in which the epigastrium is distended, and fruitless attempts are made to evacuate the stomach, the necessity of immediately using the stomach-pump is apparent from a simple survey of the situation. It is obvious that when this remedy is employed sufficiently early the fatal catastrophe of rupture of the stomach may be prevented. Lefèvre explained the importance of this indication more than thirty years ago, and recommended in the warmest terms that the stomach should be evacuated by the pump, and afterwards washed out with an emollient decoction.

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traction of the œsophagus." At all events, no actual vomiting of the ingesta occurred, and the ultimate result of the fruitless attempts to discharge them was a rupture of the stomach.

DISEASES OF THE INTESTINES.

Introductory Remarks on the Anatomy of the Parts.

Position of the Intestines.

Commencing at the pylorus, the first portion of the small intestine is the duodenum, which is about thirty ctm. in length. It consists of three parts formed by two curvatures, the pars horizontalis superior, the pars descendens, and the pars horizontalis inferior.

While the first part, as Braune especially has shown in his recent work on this subject, follows the movements of the stomach, the pars descendens is firmly bound by the ligamentum duodeno-renale to the right kidney lying behind it, and the pars horizontalis inferior by connective tissue to the subjacent aorta and inferior cava; these two portions are consequently comparatively immovable, and when distended must exert pressure upon the great vessels mentioned. The pars horizontalis superior is entirely covered by peritoneum, the pars descendens on its anterior and lateral surface, and the pars horizont. inf. only on its anterior surface.

Within the horse-shoe bend, the convexity of which is directed towards the right, is included the head of the pancreas, and into the pars descendens duodeni opens the common excretory canal of the choledoch and pancreatic ducts.

The manner in which this canal opens into the duodenum is of importance in reference to the genesis of catarrhal icterus, and will be described more fully when we come to consider the structure of the duodenum.

The duodenum is followed directly by the ileum (including under this general term the upper portion, which still retains occasionally the special name of jejunum), which, as its name indicates, appears bent into numerous convolutions. It forms nearly four-fifths of the total length of the small intestine, occupies the greater portion of the abdominal cavity, and, moreover, dips down between the organs of the pelvis. The greater portion of its convolutions are situated between the umbilicus and the symphysis, extending to the right and left as far as the lateral boundary of the anterior abdominal wall, while only a very small portion of the convolutions are in contact with the posterior abdominal wall on the right of the inferior lumbar vertebræ. (See fig. 2, p. 115.)

Beyond the small intestine lies the *large intestine*, whose long axis, however, is not directly continuous with that of the small intestine, but intersects it at almost a right angle. Since the ileum opens upon the lateral surface of the large intestine at a slightly acute angle somewhat above the very commencement of the colon, the portion of the bowel situated below the point of opening is thus naturally separated from the other portions of the large intestine. The cæcum is throughout its whole extent surrounded by a serous layer, and lies with the posterior duplicature of the latter—the meso-cæcum—upon the fascia of the right iliacus internus muscle. Below the cæcum is the *appendix vermiformis*, about seven ctm. in length, which arises from the lateral surface of the cæcum, and may occupy various positions, but *usually* extends over the psoas muscle towards the true pelvis, so that in inflammations in the neighborhood of the appendix the region along the right Poupart's ligament presents morbid changes on percussion and palpation.

Above the entrance of the ileum into the cæcum rises the colon with its well-known horse-shoe shape, which has led to a division into the ascending, the transverse, and the descending colon. These parts are connected with each other by two flexures—the right and left flexures of the colon.

The *ascending colon* and the *descending colon* are firmly bound by connective tissue to the quadratus lumborum muscle and the kidney on each side, so that on the one hand inflammatory processes in these portions of the intestine may extend to the structures referred to, and on the other hand diseases of the kidney and muscle may involve the intestine, *e. g.*, a renal abscess may burst directly into the colon. This event occurs *without injury to the peritoneum*, because the latter covers only about two-thirds of the circumference of these two divisions of the colon, while their posterior lateral surface, *i. e.*, the portion of their walls which lies over the quadratus lumborum and the kidney, is uncovered by a peritoneal layer. On the other hand, the *transverse colon* is completely covered by peritoneum, and is moreover provided with a true mesentery, the meso-colon, which, being connected with the greater omentum, permits a greater mobility in this portion of the colon than is possessed by the ascending and descending portions. Usually the transverse colon runs directly across from right to left, its upper border extending as high as the greater curvature of the stomach and nearly as low as the level of the umbilicus. (See fig. 1. p. 114.) Its comparative mobility and its distention by ingesta may, however, give it a variety of positions. I have lately seen a case (Berichte aus der Jenaer medic. Klinik, S. 41), in which the distended colon pushed the apex of the heart some two ctm. to the left, and completely replaced the dulness of the spleen and liver, the latter organ particularly being turned backwards upon its transverse axis. At other times the colon may sink below the umbilicus, or may even lie in several convolutions.

At the *flexures*, where the transverse colon passes on the right side into the ascending, and on the left side into the descending colon, the passage of ingesta seems to encounter considerable delay. This explains the fact that in inflammatory processes, particularly dysentery, the chief foci of disease are found at the flexures, and also shows the advisability of giving special attention to these places by percussion and palpation in cases of constipation.

From the crest of the left ileum onwards the colon consists of a large convolution occupying the left side of the body, entirely surrounded by peritoneum, and called the *sigmoid* or iliac *flexure*, or the S Romanum. This portion of the large intestine, from its being provided with a long meso-colon, is more mobile than the preceding part, so that when much distended it may displace the convolutions of the small intestine lying in front of it, extend upwards to the abdominal wall near the umbilicus, and thus become accessible to palpation. Usually, however, the impaction of the sigmoid flexure with fæces cannot be discovered by palpation, but only by percussion, which gives a dull note to the left of the spine just above the crest of the ilium. (See fig. 2. p. 115.)

As a rule, the sigmoid flexure consists of *a single* convolution, which is composed of a descending laterally situated limb, and a median ascending limb, which at last sinks down over the left psoas muscle into the true pelvis. This latter limb, when very much distended, may, in my experience, exert pressure upon the subjacent psoas muscle, and on bending the left thigh may even excite severe pain, which is at once and permanently relieved by completely emptying the bowel.

The final subdivision of the intestines, situated immediately below the sigmoid flexure, is the *rectum*. It begins at the sacral promontory, and in its general direction follows that of the sacrum and coccyx, making therefore at first a bend, with the concavity directed forwards—the so-called "sacral curve"—and afterwards turning backwards over the tip of the coccyx in a direction the reverse of the first curve—that is, with the concavity presenting backwards ("perineal curve").

Besides these two longitudinal curves, the rectum presents also several lateral curves, which are, however, of but little topographical importance, because, as Henle suggests, they are probably obliterated during the contraction of the longitudinal muscular layer of the rectum. Only a portion of the rectum, the uppermost section, is entirely surrounded by *peritoneum;* this portion is also provided with a mesentery, the meso-rectum, which, especially when it has become elongated by frequent distentions of the bowel, allows the latter a certain degree of mobility. The middle portion is covered with peritoneum only anteriorly and laterally, while the portion from the tip of the coccyx to the anus is wholly free from peritoneum, and is supported by the levator ani, which closes the outlet of the pelvis. While the posterior wall of the rectum, after it has lost its peritoneal covering, lies directly upon the bone, the anterior wall is in contact with the other pelvic viscera—in the male with the bladder, seminal vesicles, and prostate; and in the female with the vagina. Morbid conditions of these organs may, therefore, be palpated per rectum, operated upon, etc.; while, on the other hand, morbid conditions of the rectum, *e. g.*, fæcal impaction, may give rise to functional derangements in these organs.

Structure of the Intestines.

The wall of the *small intestine* is composed of the same membranes as that of the stomach—a *serosa*, a *muscularis* containing longitudinal and circular fibres, between which lies the plexus myentericus of Auerbach ; and a *mucosa*, the structure of which is so peculiar as to require a more detailed description.

The muscular and mucous coats are connected with each other by a delicate nervea, which is divisible into two portions—the external one composed of tense connective tissue, the internal being of loose texture, closely adapted to the folds of mucous membrane, and containing numerous vessels, nerves, and especially ganglia. The muscularis mucosæ is composed mainly of longitudinal fibres, which extend into the intestinal villi, and surround the intestinal glands. Externally it is in contact with the nervea, internally with the *propria*, which consists of reticular connective tissue, and is characterized by the abundance of its lymphoid cells. The mucosa presents elevations which are known as the "intestinal villi," and, on the other hand, the glands dip down into it.

The *villi*, which are most numerous in the upper part of the small intestine, and are about 0.5 mm. in length by 0.15 in breadth, are provided with one or more central chyle-canals, which pass downwards into a flat, widespread network of lymphatics (see further on). In addition to these canals, and the epithelium to be more fully described subsequently, the villi are also composed of blood-vessels, which form a reticulum around the lymph canals, and of bundles of smooth, muscular fibres, which, arising from the muscularis mucosæ, ascend longitudinally into the villi. The contraction of these fibres throws the villi into numerous transverse folds, and propels the contents of the vessels of the villi.

As the intestinal villi form elevations above the surface of the mucous membrane, so, on the other hand, numerous depressions are found in the form of the utricular intestinal glands. These glands, which are known as the *crypts of Lieberkühn*, resemble the gastric follicles, except that they are shorter, and are provided with a short, columnar epithelium, broadened at the base. In the duodenum are found also the *glands of Brunner*, in diminishing numbers from above downwards.

These glands differ essentially, both in position and form, from the crypts of Lieberkühn. In the first place, they do not lie, like them, with their bases above the muscularis mucosæ, but underneath it, in the nervea; they possess, moreover, a botryoidal structure of the size of a poppy or hemp seed, with separate lobules, intricately tortuous canals, and a common, excretory duct. Moreover, the epithelium lining the utricles and canals has been shown by the investigation of Schwalbe to differ from that of the Lieberkühnian follicles. It consists of columnar cells, with a distinct basal process and minute intercellular canals, and, in the granulation and cloudiness of the cell-contents, most nearly resembles the epithelium of the pyloric glands.

In addition to these glands, the intestinal mucous membrane contains also a few glandular structures of a lymphoid nature, the glandulæ congoblatæ (Henle), in the form of the *solitary* and *Peyer's* follicles. These follicles originate in an aggregation of the above-mentioned lymphoid cells, which pierce the tunica propria. The follicles are accordingly found to be composed of a fine, reticular tissue, in which are imbedded a large number of closely compacted lymph cells.

The number and size of the solitary follicles vary considerably in different individuals. Sometimes scarcely any indications of them are to be found, in other cases they form nodular elevations on the mucous membrane. They may be distributed either irregularly or at regular distances from each other, or they may be collected together in small groups, forming transitions to Peyer's patches. The latter, the aggregated glands (glandulæ agminatæ), are composed of numerous follicles, as many as sixty and upwards. They are first met with in the lower portion of the small intestine in varying numbers (twenty being the average), and are always situated opposite the attachment of the mesentery; they are circular or elliptical in shape, and are constructed in the same way as the solitary follicles.

The microscopic structure of the follicles, and especially their neighborhood to the lymph canals, prove almost conclusively that these bodies are merely small lymphatic glands interspersed in the mucosa.

The Lieberkühnian follicles and the intestinal villi are absent at those parts of the mucous membrane where the summits of the agminated glands are met with, for the reason that the former are pressed aside by the latter, and are, therefore, arranged around the latter like a coronet. These parts, as well as all the rest of the mucous surface of the intestines, are uniformly covered by an *epithelium*, which constitutes the most internal layer of the mucous membrane.

The epithelium is composed of columnar and *cup-shaped cells*, the latter probably being intimately connected with the formation of mucus. When first discovered, they were supposed to take part in the process of absorption in the small intestine; but this view has of late been justly discarded.

The *columnar cells* present on their free surface a finely striated border running parallel to the long axis of the cells.

The mucous membrane of the small intestine is thrown into *transverse folds* (plicæ conniventes Kerkringii), which begin in the descending portion of the duodenum, diminish towards the end of the ileum, and finally entirely disappear. When the intestine is relaxed they overlie each other in an imbricated manner, and can be unfolded only after the removal of the external tensely-stretched layer of the tunica nervea.

In the middle of the descending portion of the duodenum, upon its posterior wall, the regular arrangement of the transverse folds is interrupted by a longitudinal fold formed by the opening of the ductus choledochus into the intestine. This elevation of the mucous membrane is explained by the fact that the bile duct runs underneath the mucous membrane for some little distance before it finally empties into the bowel. The opening at the extremity of this ridge is marked by a papilla, which is covered from above downwards by a fold of mucous membrane (diverticulum Vateri).

The *blood-vessels* of the small intestine arise almost exclusively from the superior mesenteric artery, and pass into the superior mesenteric vein, to empty finally into the trunk of the vena portæ.

The *superior mesenteric artery* and its branches run between the two layers of the mesentery to the different portions of the intestine. The distribution of this vessel is characterized by numerous typically arranged, arched anastomoses of its branches with each other, especially by the two large anastomoses with the cœliac and inferior mesenteric arteries, which arise—the one above and the other below it—from the abdominal aorta, viz., with the cœliac artery, through the hepatic, gastro-duodenalis, pancreatico-duodenalis superior, and pancreatico-duodenalis inferior arteries; with the inferior mesenteric, through the middle colic artery, which anastomoses with the left colic artery. The latter anastomosis is much the more important of the two, and is altogether the largest in the whole body.

The transition from the arteries to the veins takes place through a fine capillary network, which pierces the follicles of the mucous membrane and surrounds the glands and intestinal villi. The vascular reticulum lies so immediately underneath the epithelium, that after the removal of the latter the vessels may very easily be injured, since the layer above them is scarcely 0.002 mm. in thickness.

The *lymphatic vessels* originate in the chyle-canals, which usually run in the central axis of the villi, and terminate in cæcal extremities. Whether these extremities are still further connected with a reticulum of very minute lymph (absorption) vessels at the summit of the villi has been a subject of constant dispute. Around Peyer's patches the lymph-vessels form an annular network. The ends of these very minute chyle-canals collect together in the propria and nervea, to form a horizontally-situated reticulum, from which arise the different efferent vessels, some of which, after piercing the intestinal wall, pass directly to the subserous lymph-vessels, while the others open first into the lymphatic network situated, with the plexus myentericus, between the transverse and longitudinal layers of the muscularis, so that the lymph current has various passages for escape (Auerbach), and is thus less likely to be obstructed.

The *nerves* of the small intestine arise partly from the abdominal plexuses of the sympathetic—the plexus cœliacus and plexus mesentericus sup.—and partly from the abdominal portion of the right vagus. The distribution of the nerves in the intestinal wall takes place through two plexuses with very numerous ganglia of microscopic size; one, the plexus submucosus, situated in the nervea, sends its fibres to the surface of the mucous membrane; the other, the plexus myentericus (Auerbach), lying between the longitudinal and transverse muscular layers of the intestinal wall, is distributed to the musculature of the intestine.

The structure of the *large intestine* is in many respects the same as that of the small intestine. Thus, its mucous membrane is likewise composed of a nervea, a muscularis, a propria formed of reticular connective tissue with lymphoid cells, and a cup-shaped and columnar epithelium, which, however, has a less characteristic border than in the small intestine. So also we find *utricular glands*, very similar to the Lieberkühnian and *solitary follicles.* In the vermiform appendix these glands are situated so closely together that this portion of the intestine may be regarded as a large agminate gland arranged in a tubular form ; elsewhere in the large intestine agminate glands are absent. The chief difference consists in the entire absence of villi upon the mucous membrane of the large intestine ; but the arrangement of the nerve- and lymph-plexuses, except so far as it is modified by the absence of the villi, is the same as that in the small intestine.

Further details are necessary only in reference to the musculature of the large intestine, the larger folds of the mucous membrane, and the distribution of the vessels, the latter bearing an important part in certain affections of the large intestine.

The *mucous membrane*, when the intestine is contracted, lies in longitudinal folds, which disappear on distention of the bowel. In the latter condition, however, there still remain the *plicæ sigmoideæ*, which are peculiar to the large intestine.

These folds are composed of transverse horizontal elevations of the entire intestinal wall, each of which occupies only about one-third of the circumference of the intestine. This limitation is due to the fact that the colon is traversed throughout its whole extent by three longitudinal bands (*tæniæ coli*), formed by a thickening of the longitudinal muscular fibres, half an inch in breadth, and giving rise to three depressions on the exterior of the intestine and internally to three projections of its wall. The three longitudinal furrows lying between the bands and formed by the inner surface of the intestinal wall are traversed in a horizontal direction by the sigmoid folds, and are divided by the latter into numerous *sacculi* (*haustra coli*), which, when the colon is much distended, project upon the external surface of the bowel in the form of bladders, separated from each other by the transverse constrictions. The sharp curved edges of the narrow crescentic plicæ sigmoideæ are not usually all situated upon the same plane, but alternate in their elevations, so that Luschka's supposition, that when the intestinal wall contracts, they act like pairs of shears to cut up the fæcal masses into so-called scybalæ, is probably erroneous; still they may be regarded as assisting to prevent, in some degree, the falling back of the more solid fæcal masses as they are propelled upwards against the force of gravity.

Besides these, which are the principal folds upon the inner surface of the intestinal wall, there are others of a special character at particular parts of the large intestine.

At the opening of the vermiform appendix into the cæcum is usually placed a duplicature of mucous membrane, which more or less blocks up the opening, and which, in Gerlach's experience, appears to become rudimentary in advanced years, thus explaining the diminished frequency of fæcal impaction in the vermiform process and of perforations of its wall in old persons.

At the opening of the small intestine into the large intestine is situated a double fold, formed by the protrusion of the former into the lumen of the latter, and called the *valvula coli*, or valve of Bauhin. This valve is composed of the mucous membrane and the layer of circular muscular fibres of the small intestine, while the layer of longitudinal fibres and the serosa are directly continuous with the wall of the large intestine.

Finally, upon the mucous membrane of the rectum, besides the folds which are easily effaced by the stretching of the gut, there is also found a permanent, ineffaceable transverse falciform fold (*plica transversalis recti*), situated from six to nine ctm. above the anal orifice, usually anteriorly, and on the right side. *It should be the rule, therefore, in introducing bougies, etc., into the rectum, to direct the instrument to the left and posteriorly.*

In the most inferior portion of the rectum, where the mucosa is devoid of glands and is transitional between the skin and mucous membrane, project from five to eight small longitudinal folds, the *columnæ Morgagni*, between which fæcal particles may be retained, sharp bodies be caught, and ulcerative foci be readily formed.

The *musculature* of the large intestine, like that of the rest of the digestive tract, is composed of an external longitudinal and an internal circular layer of fibres. The former consists of the above-mentioned muscular bands—the tæniæ coli—which unite at the beginning of the rectum. The circular fibres increase in thickness inferiorly, and at the anus form a complete girdle, the sphincter ani internus, which is also joined at the extremity of the rectum by another unstriped muscle, the musculus recto-coccygeus. Finally, the striped muscles, connected with the rectum, are the levator ani and the sphincter ani externus

Vessels of the large intestine:

The arterial supply is derived from the superior and inferior mesenteric arteries, the latter of which gives off the superior hemorrhoidal to the sigmoid flexure and the posterior wall of the rectum. The superior hemorrhoidal communicates with the middle hemorrhoidal derived from the hypogastric, and with the inferior hemorrhoidals from the common pudendal. The capillaries of these vessels surround the intestinal glands, and around the orifices of the latter form a coronal reticulum, from which in turn arise the venous radicles. The minute distribution of the vessels in the large intestine is similar, therefore, to that which is found in the gastric mucous membrane. The *venous* trunks correspond in course and name to the arteries above mentioned; thus, the veins of the colon empty into the superior and inferior mesenteric veins; in the rectum we have the inferior and middle hemorrhoidals, which empty into the internal iliac vein, and the superior hemorrhoidal which empties into the inferior mesenteric, and thus into a branch of the portal vein, forming *an anastomosis of great pathological importance between the circulation in the chylopoetic viscera and that of the rest of the lower half of the body.* The plexus formed by these different veins around the rectum (plexus hemorrhoidalis) is situated in the tunica nervea, and in the main runs parallel to the long axis of the intestine. The inferior veins, after piercing the external sphincter muscle, form around the anal orifice a subcutaneous network, the engorgements of which give rise to what are known as *external hemorrhoids.*

Physiological Considerations.

The chyme, after escaping from the stomach through the pylorus, becomes mixed with the *secretions of the liver, pancreas, and secernent glands of the intestines.* The intestinal juice is supplied almost exclusively by the Lieberkühnian follicles, and only to a very small extent by the duodenal glands of Brunner. All[1] of these secretions take an important part in the processes of digestion and absorption which have been energetically begun in the stomach. It is, of course, unnecessary to enter here into full details in regard to their composition, formation, excretion, and action, and in the following remarks these points will be touched upon only so far as a knowledge of them is essential to an understanding of the diseases of the intestinal canal.

In the first place, the secretion of the liver—the *bile*—which is an extremely important secretion for the processes of digestion and absorption in the intestines, contains, as its *principal ingredients*, the potash and soda salts of the *biliary acids*, the glycocholic and taurocholic acids, *cholesterin* and the *biliary coloring matters*, particularly the bilirubin, which is a derivative of the coloring matter of the blood, and seems to be identical with hæmatoidin. It gives to the bile its yellow color; in contact with the acids of the ingesta in the duodenum it becomes converted into biliverdin, and thus the bile acquires a green tinge. The *reaction* of the bile is neutral or feebly alkaline. The secretion is formed by the liver cells out of the material brought to them by the hepatic artery and portal vein; it takes place continuously, and does not seem to be dependent, at least not directly dependent, upon the influence of the nerves. When once formed, it is propelled by the *vis a tergo* of the newly-forming secretion into the bile ducts, thence into the hepatic duct, and is finally discharged along with the bile from the gall-bladder through the choledoch duct into the duodenum. The secretion is discharged more copiously during digestion, a fact which may be explained partly by the mechanical pressure which the contents of the stomach exert upon the liver after a meal, and partly by the contraction of the gall-bladder, and probably also of the bile ducts, which occurs simultaneously with the peristalsis. Furthermore, as the acid chyme from the stomach passes over the orifice of the choledoch duct it excites a reflex contraction of the biliary passages. The escape of the bile from the liver is also favored by the compression of the organ during inspiration. The *quantity* of bile discharged varies according to the time which has elasped since the previous meal; it begins to increase after taking food, and attains its maximum in about seven hours. The quantity of the bile is influenced also by the quality of the food, being greater when the food consists of albuminous substances, while fats produce no alteration. According to the observations made by Joh. Ranke and von Wittich, in cases of persons with biliary fistulæ, *the daily discharge of bile* amounts to from twelve to thirty-two ounces. The *chief functions* of the bile consist in the prevention of putrefactive processes in the intestinal canal, in the conversion of starch into sugar, especially in the precipitation of the syntonin and pepsin (as described more fully on page 125), whereby the digestive action of the pepsin is suddenly arrested in the duodenum; finally, and most important of all, in the part it plays in promoting the absorption of fat. This property, which is possessed in a still more marked degree by the pancreatic juice, is probably to be explained by the fact that the soaps formed by the combination of the alkalies of the bile with the free fatty acids of the chyme possess the power of rapidly emulsifying fat. Furthermore, the absorption of fat is favored mechanically by the presence of bile in the capillary absorbent vessels, into which the bile readily passes, since experiment has shown (von Wistinghausen) that fat readily ascends in capillary tubes, the walls of which have been moistened with bile, but fails to do so if the capillary wall is moistened with water or watery solutions.

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[1] Hitherto it has been impossible to obtain the secretion of Brunner's glands by itself, and therefore nothing positive has been ascertained in regard to its composition and action.

The *pancreatic secretion* has a very alkaline reaction, and contains, as specific ingredients, *leucin* in very large quantities (tyrosin in small amount), *ferments*, and particularly pancreatine, together with lactic acid, fats, and other substances of subordinate importance. *By means of its ferments and its richness in soda salts, the pancreatic secretion is able to exert the most far-reaching digestive powers.* It converts starch into sugar with an energy surpassing that of the saliva; like the bile, it saponifies and emulsifies fats—this being its main function; and, finally, it exerts a digestive action upon albuminous substances and the glutens, converting them into peptones and ultimately into leucin, tyrosin, and the fæcal-smelling indol. The *formation of the pancreatic juice* undoubtedly takes place in the cells of the pancreatic gland, and is accompanied by swelling and reddening of the organ. The discharge of the secretion seems to be excited by the gastric nerves, so that the acid gastric juice and the alkaline pancreatic juice are poured out simultaneously. The quantity of the latter secretion has never been ascertained.

The *intestinal juice*, secreted by the Lieberkühnian follicles throughout the entire length of the intestines, is obtained in a pure state by isolating a loop of intestine in the manner practised by Thiry. Obtained in this way, the secretion is a thin, strongly alkaline fluid. Its digestive power is certainly much less active than that of the pancreatic juice; but its effects are not yet definitely determined. It is certain, however, that crude fibrin is dissolved by it, and that it produces peptones which, according to my investigations,[1] are analogous in their reactions to the peptones produced by the gastric and pancreatic juices. On the other hand, albumen coagulated by heat or acids and boiled fibrin, do not appear to be attacked by the pure juice. Furthermore, the intestinal juice of dogs does not under all circumstances convert starch into sugar or emulsify fats, but I have always found it to possess the power of *converting cane into grape sugar.* Human intestinal juice, however, as shown by Busch's[2] experiments in his remarkable case of fistula of the small intestine, is probably able to partially dissolve pieces of albumen and meat, and to convert starch into sugar; but Busch was unable to discover any conversion of cane into grape sugar. That this juice actually does possess saccharifying properties is in the highest degree probable, from the experiments of Kühne with cholera stools, which were found to convert starch into sugar energetically. There was no evidence in this case of any pancreatic juice in the evacuations; while, on the other hand, the saccharifying power was too marked to be accounted for by an admixture of swallowed saliva. The digestive action of the intestinal juice, like that of the other digestive juices, depends upon the presence of ferments. The *quantity* secreted during digestion is doubtless considerable, since we know that the usually almost entirely quiescent secretion can be abundantly increased by mechanical and chemical irritants, such as the action of acids, etc.

The digestive changes effected by the secretions above described are carried on chiefly in the small intestine, and there, also, most of the products of digestion are absorbed. This is the case particularly with the fats, which, after having been saponified and emulsified by the pancreatic juice and the bile, are absorbed by the villi. After the use of fatty food, the chyle-vessels present a white, milky appearance, in consequence of their being filled with chyle, which is made opaque by the suspended fat. The absorption of the fat through the villi is effected chiefly by the contraction of the muscles of the villi. When irritated, the villi contract in a transverse direction, and exhibit a vermicular wrinkling, like that of an "œstrus-larva." By this means the contents of the villi are forced downwards into the chyle- and blood-vessels. As the muscles relax, the chyle is prevented from returning by the valves which exist in the minute chyle-vessels, and, the internal pressure being thus removed when the villi again become erect, a renewed process of absorption takes place. The absorption of fat is usually completed by the time the chyme has reached the lower part of the small intestine; still, under special circumstances, the fat may be absorbed even after it has passed into the large intestine (see below).

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[1] Med. Centralblatt. 1868. S. 289.

[2] Virchow's Archiv. XIV. S. 140 ff.

The *sugar*, most of which is formed from the starch, under the influence of the pancreatic juice, and probably also the *dextrin*, are likewise absorbed in the small intestine; so also the *lactic acid salts* arising in the small intestine from the combination of the alkalies of the intestinal juices with the lactic acid produced in the stomach by the fermentation of the carbohydrates. Most of the sugar, like the fat, is undoubtedly absorbed by the chyle-vessels, as is shown by the fact that the portal vein never contains more than a small amount of sugar. At the same time there can be no question that at least a small portion of the readily diffusible saccharine and saline solutions are absorbed, not by filtration in the way described, but by a simple diffusion into the blood-vessels. Even of the fat, small quantities are absorbed by the blood-vessels in the form of saponaceous solutions, as is shown by the blood always containing soap, and by the experimentally-demonstrated fact that, when soap is fed to an animal, by far the greater portion is absorbed (Radziejewski).

It is in the highest degree, probable, therefore, that the carbohydrates, as well as the fat, are absorbed in both ways—that is, by both the lacteals and the blood-vessels, though undoubtedly chiefly by the lacteals. The same is true of the absorption of the *albuminates* and their derivatives. The hypothesis long ago advanced by Brücke, that albuminous substances are absorbed as such by the chyle-vessels before they have been converted into peptones, cannot be disputed at the present day; but, admitting this fact, it is difficult to see why the readily diffusible peptones should not also be absorbed by the blood-vessels like other perfect solutions. Nor is there any reason to suppose that the peptones, after having passed into the blood, are merely converted into urea or the intermediate products of nitrogenous metamorphosis. On the contrary, the absorbed peptones play an important part as nutritive material (Plósz, Maly), being reconverted into albuminous substances—a synthesis of which there are still other instances in the process of assimilation.[1] The absorption of peptones takes place through both the lacteals and the blood-vessels; while the albuminates, which have not been converted into peptones, are probably taken up exclusively by the lacteal vessels, in which they can be directly demonstrated.[2]

All these absorbent processes undoubtedly take place chiefly in the small intestine, which, through its villi, is provided with an anatomical mechanism eminently adapted for absorption. The final share in this process devolves, however, upon the large intestine, as is shown by the fact that the fluid chyme, after reaching this part of the digestive tract, becomes more and more inspissated. The probability that the large intestine plays a more important part in the absorbent process than is commonly supposed, is supported also by the fact that, in dogs with a fistula of the colon, the food escapes through the opening as early as an hour and a half after food is taken, while, as is well known, the food usually tarries in the large intestine for at least twenty times this period.

All the digestive processes which take place in the large intestine are, however, of a merely *secondary* character, and are the result, partly of the continued action of the pancreatic juice, and partly of the continued transformation of the albuminates into peptones. The carbo-hydrates are here more completely fermented, with formation of sugar and especially lactic acid, to the presence of the latter of which is due the acid reaction of the contents of the large intestine, in contrast with the alkaline reaction of those of the small intestine. Furthermore, the transformation of the albuminates proceeds beyond the formation of peptones, leucin, and tyrosin, to the production of the fæcal-smelling indol—a transformation closely allied to what takes place in ordinary putrefaction of the albuminates. The large intestine contains also a large amount of *gases*, which are to be regarded as the result of this spontaneous decomposition in the large intestine, and which consist of carbonic acid gas, hydrogen, nitrogen, light carbonetted hydrogen gas, and almost always sulphuretted hydrogen. To the latter gas, in connection with the volatile fatty acids and the offensive indol, is due the repulsive feculent odor of the contents of the large intestine. Finally, the remains of the unabsorbed ingesta accumulate in the rectum, and form the *fæces*.

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[1] *Vid.* Hermann, Physiologie. 5. Aufl. S. 169.

[2] *Brücke*, Vorlesungen. 1. S. 338.

The following are the principal ingredients of the fæces: Cellulose, chlorophyll, small quantities of unabsorbed albuminates, lactic acid and grape sugar, wholly undigested particles of food, such as pieces of meat, starch grains, fats, etc., besides excretin, cholesterin, choloidic acid, dyslysin, stercobilin, and, when the food has passed rapidly through the intestinal canal, also the salts of the biliary acids, bilirubin, and probably unaltered pancreatic juice. Among other ingredients may also be found lime- and magnesia-soaps, volatile fatty acids, various salts, including the ammoniaco-magnesian phosphate, and not unfrequently fungi.

It is evident, from the preceding considerations, that *the large intestine possesses no specific digestive function*—at least, nothing more than a feeble digestive action on the part of the secretion of the Lieberkühnian follicles (see above). This absence of digestive power in the large intestine has recently been directly confirmed by the experiments of Czerny and Latschenberger, in the case of a patient with a fistula of the colon. They found, however, that the human large intestine possesses the power of absorbing fluid solutions of albumen, emulsions of fat, and starchy mixtures.

That the large intestine under certain circumstances is able to absorb fat, was discovered many years ago by Kölliker;[1] he introduced oil into the rectum of a young cat, and found the epithelium of the large intestine filled with fat. Eimer[2] found that in the frog, while small quantities of fat remained unabsorbed by the large intestine, absorption took place when large quantities were used;[3] and I have myself found that, in the dog, fat, although usually unabsorbed by the large intestine, is taken up if the fat be emulsified by the addition of pancreatic juice.

As regards the clinical application of these results to the *composition of nutritive enemata*, future experience must decide whether simple solutions of albumen, with or without the addition of fat and starch, can accomplish as much, and are practically as available as the enemata, composed of meat or fat with pancreatic juice, recommended by me, and since then repeatedly tried by others.

The propulsion of the chyme in the intestines is effected by the so-called *motus peristalticus*, which manifests itself in the well-known "vermicular" movements of the intestine. These movements are generated in a reflex manner by the irritations of the intestinal mucous membrane. The nerve centres immediately concerned in the production of the peristalsis are situated in the ganglia of the intestinal wall, as shown by the fact that these movements still continue in an exsected portion of the intestine. More remotely concerned are the vagus and splanchnic nerves. It has been well established by Pflüger that *irritation of the splanchnic nerve arrests the movements of the intestine*, while, on the other hand, the vagus has probably a motor action upon the peristalsis. The discharge of nerve force by which the contractile movements are ultimately excited seems to be determined by the *venosity* of the blood circulating in the wall of the intestine, or, in other words, by a lack of oxygen. Saturation of the blood with oxygen checks peristalsis, while asphyxia increases the movements. At present, however, there is no one theory which satisfactorily explains all the phenomena of the peristaltic *movements.*

The peristaltic movements take place always in *one* direction. Anti-peristaltic movements, although we are compelled to infer their occurrence in certain pathological conditions, are *never* observed in the normal animal. Sanders, however, has seen antiperistaltic movements, limited to the colon, in a case of diarrhœa, in which the movements in all other parts of the intestine took place peristaltically.

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[1] Würzburger Verhandl. Bd. VII. S. 174.

[2] Virchow's Archiv. 48. S. 119. ff.

[3] Deutsches Archiv für klin. Medicin. 1872. Bd. X. S. 37.

The peristalsis is particularly active in the small intestine, so that normally the chyme is propelled into the large intestine very rapidly (see above). In the colon, on the other hand, the peristalsis takes place very slowly; hence the very long delay of the fæces in this portion of the intestines. The reflux of the contents of the colon into the ileum is entirely prevented by the valve of Bauhin. Finally, in the sigmoid flexure and rectum we find the fæces proper. Their discharge is effected, as is well-known, by the aid of abdominal pressure. During the act of defecation the straining probably produces a temporary accumulation of carbonic acid in the intestinal vessels, and thus increases the peristalsis; at the same time the contents of the bowel are directly compressed and expelled by the act of straining, the resistance of the rectal sphincters being overcome by the pressure of the descending fæcal mass, while according to Luschka the levator ani draws the end of the rectum forwards and upwards, and the posterior wall of the bowel glides over the fæcal column.

Enteritis. Catarrhus Intestinalis. Inflammation of the Intestines. Intestinal Catarrh.

Acute and Chronic Intestinal Catarrh. Inflammation of the Duodenum. Jejunitis. Ileitis. Typhlitis. Perityphlitis. Proctitis. Periproctitis. Intestinal Catarrh of Childhood.

For the older literature see *Plouquet*, Lit. dig. under Diarrhœa, etc.—*Celsus*, Lib. IV. 19.—*Morgagni*, De sedibus, etc. Ep. LXV. Abs. 5.—*Hennings*, Kennzeichen und Heilart der Entzündungen des Magens und der Gedärme. Kopenhagen. 1795. —*Pemberton*, Diseases of the Abdominal Viscera. Lond. 1807.—*Broussais*, Histoire des phlegmasies ou inflammations chroniques. Paris. 1822.—*Billard*, De la membrane muqueuse gastro-intestinale, etc. Paris. 1825.—*Abercrombie*, Pathol. and prac. researches on diseases of the stomach and intestinal canal. 1828.—*Lesser*, Die Entzündung and Verschwärung der Schleimhaut des Darmkanals. 1830.—*Eisenmann*, Die Familie Rheuma. Band III. S. 331.—*Mayer*, Die Krankheiten des Zwölffingerdarms. Düsseld. 1844.—*Duclos*, Ueber Zahndiarrhœ. Bull. de thér. 1847. Schmidt's Jahrb 58. S. 54.—*Hauner*, Mittheilung aus dem Kinderspital zu München. Deutsche Klinik. No. 9. 1851.—*Schürmans*, Ueber die Enteritis der Kinder. Presse méd. 1851. Schmidt's Jahrb. 75. S. 190.—*Rilliet*, Ueber einige Krankheiten des Verdauungskanals während der ersten Kindheit. Gaz. de Paris. 1853. Schmidt's Jahrb. 80. S. 61.—*Virchow*, Historisches, Kritisches, und

Positives zur Lehre der Unterleibsaffectionen. Virchow's Archiv. V. Heft 3. S. 348. 1853.—*Boens*, Verengung des Mastdarms durch Neubildung in dessen Umgebung. Journ. de Brux. 1855. Schmidt's Jahrb. 90. S. 315.— *Willigk*, Sectionsberichte, etc. Prager Viertelsjahrschrift. XIII. 2. 3. 1856.—*Bourdon*, Verengerung der Ileocoecalöffnung. Union méd. 57. 1856. Schmidt's Jahrb. 96. S. 204.—*Habershon*, Pathological and practical observations, etc. London. 1857. Ibid. 100. p. 262.—*Geist*, Klinik der Greisenkrankheiten. Erlangen. 1857–1860.—*Le Barillier*, Ueber die Enteritis der Säuglinge. Journ. de Bord. 2. Ser. V. 1860–61. Schmidt's Jahrb. 111. S. 64.—*Luzsinky*, Dritter Jahresbericht, etc. Journal für Kinderkrankheiten. 1859. S. 254. 1861. S. 229.—*Poland*, Contusion of the abdomen. Guy's Hosp. Rep. 3. Schmidt's Jahrb. 105. S. 76. 1860.—*Bouchut*, Pract. treatise on the diseases of children, etc., London, 1855, with a full digest of the literature of the subject.—*Winternitz*, Hydriatische Behandlung der Diarrhœ im Kindesalter. Jahrb. für Kinderheilkunde. VIII. Heft 3 u. 4. 1867.—*F. Siredey*, Note pour servir à l'étude des concretions muqueuses membraniformes, etc. Union médic. 1869.—*Courval*, Aehnl. Beobachtung Canstatt's Jahresb. 1869. S. 132.—*Oppenheimer*, Versuche über Hydrotherapie bei Diarrhœ der Kinder. Bayer. Aerztl. Intelligenzbl. 1869. No. 24.—*Simon, J.*. Notes pour servir à l'histoire de quelques diarrhées specifiques. Archives génér. de méd. 48. 180. 1870.—*Sutton*, A burn; death from profuse diarrhœa, etc. Brit. med. Journ. 1870.—*Da Costa*, Membranous enteritis. Americ. Journ. of the Medical Sciences.—*Whitehead*, Mucus disease. Brit .Med. Journ. 1871. S. 149.—*Fraentzel*, Enterotomie bei Ileus. Virchow's Arch. 49. 164.—The recent works of *Bamberger*, *Gerhardt*, *Steiner*, *Canstatt*, *Wunderlich*, *Henoch*, *Valentiner*, etc.

In the same way as dyspepsia was classified among the inflammations of the stomach, so diarrhœa might with equal propriety be included among the inflammations of the intestines—in fact, it was expressly maintained by Broussais that diarrhœa could not occur without an inflammation of the intestinal mucous membrane. This view is, however, opposed by the common experience, that a sudden attack of diarrhœa may be caused by mental emotion and other nervous influences. A satisfactory explanation of such cases is impossible in the present state of physiological science, because our knowledge of the innervation of the intestines, as it is concerned in the production of the peristaltic movements, is too indefinite, while it is quite useless to resort to such explanations as an irritation of the vagus, a temporary paralysis of the mesenteric nerves, or similar possible causes. At all events, in this form of diarrhœa the attack rapidly subsides, and, just as after the administration of a simple laxative, only one or two loose discharges occur without any subsequent evidence of an intestinal lesion. Such deviations from the ordinary movements of the intestines may probably, therefore, be regarded partly as still lying within physiological limits, and partly as neuroses of the intestine, as, for example, the diarrhœas of hysterical persons.

In the following chapter, therefore, we shall confine ourselves to the consideration of those pathological changes and morbid symptoms, which are connected with conditions that are generally acknowledged to be of an inflammatory nature.

Pathological Anatomy.

The intestinal mucous membrane in acute catarrh is more or less vividly reddened and swollen, its superficial layers occasionally œdematous, the epithelium abundantly desquamated, the cells enlarged, the protoplasm granulated, and the nuclei indistinct.

The microscopico-anatomical changes last mentioned are not, however, to be regarded as pathognomonic of intestinal catarrh, for the reason that in the intestine, just as in the stomach, post-mortem changes may take place in the inflamed tissue, particularly the epithelium, from the action of the digestive juices, which retain their powers for some time after death; while the same changes may occur in the desquamated epithelium during life when the epithelium has been subjected to a process of digestion. The presence of altered epithelium in the dejections of individuals with intestinal catarrh is in itself, therefore, no evidence of intestinal inflammation—indeed, the only circumstances under which such a conclusion could be drawn, would be, when an artificial inflammation of the intestines had been excited in an animal, and the mucous membrane had been examined immediately after death. Moreover, it should be borne in mind that all the symptoms of intestinal catarrh may be present during life without any injection of the mucous membrane after death, just as happens in the case of other mucous membranes which can be directly inspected during life.

When the injection is well marked, it is sometimes bright, sometimes dark; in extent it may be either diffuse or circumscribed, and it is especially noticeable in the vicinity of the glands. Frequently, extravasations of blood are also found. The *inflammatory exudation upon the free surface* is probably very copious, and undoubtedly contributes to the liquefaction of the fæces; but the fluidity of the discharges in enteritis is to be explained chiefly by the fact that the intestinal juices, which recent experiments have shown to be poured out in great abundance, are propelled rapidly downwards, in consequence of the active peristalsis excited by the inflammation, before the absorption of the products of digestion and the condensation of the fæces can take place. The longer the inflammation continues the greater will be the number of extravasated *pus corpuscles* mixed with the inflammatory exudation.

The *solitary follicles* and those of the *agminated glands* may become swollen, especially in the more severe forms of acute catarrh, and then appear as dull-gray vesicular prominences as large as the head of a pin, surrounded by a reddened border. If the swollen glands become necrosed, the so-called follicular ulcers are formed. The latter will be described more fully subsequently, as will be also the catarrhal ulcers which arise from small erosions at points where the epithelium has been destroyed.

In *chronic* intestinal catarrh, which is generally developed from the acute form, the chief changes are a dilatation of the veins, and a deep diffused redness mixed with a brown slaty pigmentation, especially of the villi, resulting from previous extravasations. In this form of catarrh, also, we find the mucous membrane swollen, and covered with an abundant purulent exudation, while the alteration of the epithelia is shown by the indistinctness of their nuclei and by the molecular cloudiness and fatty degeneration of their protoplasm. The solitary *follicles* are still more swollen than in acute catarrh, pigmented, and sometimes converted into ulcers. In the large intestine more particularly, the mucous membrane, or rather its submucosa, proliferates in various places in the form of papillary tumors (enteritis polyposa). Modifications in the usual anatomical picture of enteritis may also be produced by changes in the Lieberkühnian follicles. Thus the secretions may accumulate in the gland-sacs, so as to give rise to mucus-comedones with a dilatation of the glandular orifices which is visible to the naked eye; or, as in a case observed and admirably described by Virchow,[1] the gland-cysts filled with mucus may coalesce to form large prominent clusters in the mucosa, some of which retain this character, while others supply the materials for the formation of polypoid proliferations (*enteritis*, particularly *colitis*, "*cystica*" *polyposa*). When these proliferative processes are of a more diffuse character, the mucous membrane becomes thickened, and undergoes chronic induration. In other cases, especially in children, a long-continued catarrh may lead to *atrophy* of the mucous membrane, which then looks pale and yellow, and the gland-sacs seem appreciably shortened.

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[1] *Virchow*, Die Krankhaften Geschwülste. I. S. 243.

As in chronic gastritis, so also in chronic inflammation of the intestines, the *muscularis* shares in the morbid process. Sometimes the muscularis becomes hypertrophied to such a degree as to narrow the lumen of the intestine (simple stricture). This result is most frequent in the rectum, the sigmoid flexure, and at the valve of Bauhin (Bourdon), but in rare cases it is met with also in the small intestine. Atrophy of the muscularis is seen in children with chronic intestinal catarrh, and in persons suffering from wasting diseases.

Finally, in the more intense forms of the disease the *mesenteric glands* may also be enlarged.

The different portions of the intestinal tract do not manifest an equal tendency to catarrh, although the affection may occur at any place from the stomach to the rectum. The *colon* is affected most frequently, probably because irritating substances are delayed here longer than elsewhere in the intestines, and because the wall of the colon is exposed to the friction of indurated fæcal masses. This fact, together with the circumstance that the fæces begin their putrefactive changes while still in the colon, and that septic organisms probably cling more readily to the mucous membrane when it is inflamed, explains also the special frequency with which *diphtheritic* deposits are met with in inflammations of the large intestine, giving the mucous membrane the appearance as if sprinkled with bran (see below).

According as the catarrh affects different portions of the intestinal canal, special terms are used to distinguish the varieties of the disease: *duodenitis*, *ileitis*, *typhlitis* (cæcum), *colitis*, and *proctitis* (rectum).

Duodenitis is characterized by the accompanying jaundice, which results from the catarrhal swelling of the ductus choledochus, and the occlusion of its orifice; while ileitis is attended by a tumefaction of Peyer's patches, and under some circumstances by a narrowing of the ileo-cæcal valve from simple inflammatory hypertrophy.

Typhlitis, which is produced especially by fæcal impaction and stercoral calculi (*typhlitis stercoralis*), seems to be very generally connected with a relaxed condition of the intestinal muscles, which, in its turn, materially favors the retention of the fæces. If the irritating substances fail to be removed, the inflammation extends to the deeper layers of the intestinal wall, and produces ulcerations which may lead to perforation—results, which will be considered more fully under the head of Intestinal Ulcers. Similar to typhlitis in its origin and course, is the *inflammation of the appendix vermiformis.* This also very generally terminates in ulceration, which is one of the most common causes of perforation-peritonitis (see below). In other cases the vermiform canal becomes constricted or obliterated, either at its orifice or at some distance from it, so as to prevent the escape of the mucus secreted in its interior. The canal in this way becomes distended into a round sac, which may attain the size of a large fist (Virchow). The contents of this sac, as just remarked, consist at first of tenacious mucus, but are afterwards changed to watery serum, because, when the wall of the appendix becomes much distended, it also becomes thinned, its inner surface smoother, and the distribution of its vessels more superficial. In this way the escape of the watery portion of the blood is favored, and the formation of mucus is reduced to a minimum (hydrops processus vermiformis).

In *proctitis* the above-described changes are found most strikingly developed : marked swelling of the mucous membrane, blennorrhœa, branny diphtheritic deposits, ulceration, polypoid proliferations, and finally hypertrophy of the submucosa and muscularis. This hypertrophy is at times so considerable as to convert the rectum into a " thick indurated tube " firmly bound by the proliferated tissue in its vicinity. At the same time the intestine above the inflamed part is dilated and impacted with fæces, while the rectal veins, particularly those at the anus, are engorged (hemorrhoids). This venous engorgement is due, partly to the incessant irritation of the mucous membrane, and partly to purely mechanical causes, such as the impediment, presented by the conditions above mentioned, to the return of the blood from the hemorrhoidal plexus.

In each of these two forms of intestinal inflammation—typhlitis and proctitis—the inflammatory process sometimes extends beyond the intestinal wall to the connective tissue in the neighborhood of these portions of the intestine, and produces the symptoms of *perityphlitis* and *periproctitis*. Excluding the cases in which these affections arise from unknown causes, or are developed in the course of infective diseases, or result from the extension of an adjacent inflammation (caries of the vertebræ, pelvic bones, etc.), perityphlitis and periproctitis, when dependent upon intestinal inflammation, may terminate in two ways: either in an absorption of the inflammatory products followed by induration of the tissue, or the pus formed may burst, either externally or into the intestine. In the latter case, collections of pus, fæces, and necrosed shreds of tissue are formed, which communicate by fistulous openings with the affected part of the intestine (the so-called internal rectal fistulæ in proctitis). These secondary collections may rupture in their turn in other directions: the periproctitic abscesses externally in the anal region with the establishment of a complete rectal fistula, or into neighboring cavities, such as the bladder; the perityphlitic abscesses into the peritoneal cavity, into more remote portions of the intestine, or externally. In the external rupture, the final event is generally preceded by a burrowing of the pus along the connective tissue which surrounds the cæcum and ascending colon and extends directly to the rectum, kidneys, and superficial fascia of the upper part of the thigh.

Etiology.

In order to understand the frequency of intestinal inflammations, it is necessary to recall some of the anatomical peculiarities of the mucous membrane of the intestines. In the introductory remarks on anatomy it was mentioned that the network of minute blood-vessels in the villi of the small intestine is situated immediately underneath the epithelium, and that for this reason the vessels are directly exposed to the influence of irritating substances. Furthermore, the arrangement of the venous twigs in the mucous membrane of the large intestine is exactly the same as in the gastric mucous membrane (see above), so that the return of blood from the capillaries meets with a certain degree of resistance. A similar effect is produced, also, by another anatomical peculiarity of the intestinal circulation, to which Rindfleisch particularly has called attention, viz., that while the arteries in their passage through the muscularis of the intestine are surrounded by quite a strong sheath of connective tissue, the venous twigs, on the contrary, pierce the muscular coat in an exposed condition, so that every muscular contraction produces more or less interference with the *return flow* of blood. Finally, from the fact that during the process of digestion the chyle-vessels of the mesentery always contain red blood-corpuscles, it may probably be inferred (Cohnheim) that the intestinal mucous membrane is one of the regions of the body where the passage of blood-cells through the walls of the vessels takes place with special facility. It is not surprising, therefore, that the *intestinal mucous membrane should be particularly liable to inflammation*, and that even slight irritations may suffice to excite the same.

The exciting *causes* of intestinal catarrh are therefore extremely numerous, as the following enumeration will abundantly show.

Not to speak of the intestinal catarrhs which are produced by the extension to the intestinal mucous membrane of an inflammation in a neighboring organ, the biliary passages, stomach, or peritoneum, the occurrence of intestinal catarrh always presupposes one of two etiological conditions: either the *irritations* are of *abnormal* character, the mucous membrane being normal, or, for some cause, the *intestinal mucous membrane has lost its normal character*, so that even normal irritations are sufficient to excite an inflammation.

Under the *first* of these classes of causes are included all articles of food which produce severe mechanical or chemical irritation, coarse indigestible food, unripe fruit—which has been imperfectly masticated—fermenting drinks, etc.; also *foreign bodies, poisons, and medicines*, such as colocynth and other powerful purgatives. Intestinal inflammation, particularly in the rectum, seems in some cases to be produced also by a massive accumulation of worms. Another not infrequent cause of intestinal catarrh is the presence of intestinal concretions, or indurated fæces, especially above places where the intestine has been narrowed by a bend of the bowel, adhesions, etc. The latter of these causes of inflammation—fæcal impaction—not merely acts mechanically upon the intestinal mucous membrane, but also chemically by means of the putrefactive changes which take place in the retained fæces. To these putrefactive influences may be in part ascribed also the diphtheritic inflammations of the intestine, which are found in cases of pyæmia and *septicœmia*, and not unfrequently also in puerperal women (see below).

Another very common cause of intestinal catarrh is the *influence of cold*, which probably acts by driving the suddenly cooled blood upon the surface of the body into the intestines, where the irritation excites increased peristalsis and inflammation. Finally, abdominal *injuries* (contusions of the abdominal wall) may produce intestinal catarrh even of a chronic character, as is shown by Braillet's case (see Poland), in which a man, whose abdomen had been trampled upon, died several months afterwards of ileus, resulting from traumatic jejunitis.

In the *second category* of causes of intestinal inflammation, viz., when the mucous membrane has lost its normal character and the catarrh supervenes secondarily as a result of this change in its condition, are included all abnormal conditions which induce a morbid hyperæmia of the intestinal mucous membrane. Chief among these causes are the *passive congestions in the portal circulation* produced by affections of the liver or of the vena porta itself, or by tumors compressing the mesenteric veins; also the general congestions which occur in connection with *cardiac* and *pulmonary diseases*, and which, by inducing a permanent engorgement of the intestinal mucous membrane, excite a predisposition to catarrh. Such a predisposition must also be inferred in patients with *tuberculosis*, *morbus* Brightii, etc., the liability to catarrh in these cases probably arising from defective nutrition of the walls of the vessels. Whether the intestinal inflammations which occur after burns[1] are to be regarded as due to a similar cause, I shall not attempt to decide. The connection is more obvious between intestinal inflammation and *incarcerations*, *torsions*, and *intussusceptions*, because here the intestinal vessels are twisted and compressed, stases occur in the free circulation leading to passive hyperæmia of the intestinal wall, and ultimately to an intense catarrh of the mucous membrane at the affected part; while above the constricted point the accumulated fæcal masses act as a permanent source of irritation. Finally, the various *ulcerative* processes which occur in the intestinal mucous membrane—the dysenteric, the tuberculous, the cancerous, and the typhoid—like ulcerations in other parts of the body, maintain in their vicinity an inflammation which, under favoring circumstances, may extend to a considerable distance around the ulcers. The same relation holds between neoplasms of the intestine and the secondary catarrh excited by them.

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[1] In cases of burns Falk (Virchow's Archiv. 53. S. 27) has found broken-down blood corpuscles in the blood. and a depressed action of the heart, conditions which may help to explain the singular reciprocal relation between intestinal inflammation and burns of the skin.

While it has long been known that diarrhœa of a tertian or quotidian type may be produced by *malarial* poisoning, Jules Simon has recently observed several cases in which diarrhœas of years' duration, without any regular type, and sometimes accompanied by enlargement of the spleen, sometimes not, disappeared at once as soon as quinine was administered, after all other remedies for diarrhœa had been used in vain. The admixture of mucus and blood in the stools, and the fact that in one striking case the diarrhœa, although relieved by quinine, was not entirely controlled until local treatment was employed, show that malarial poisoning may occasionally be the cause of a *chronic intestinal catarrh.*

According as the exciting cause of the intestinal catarrh is of a temporary, frequently recurring or permanent character, the affection runs an *acute* or a *chronic* course.

The disease is a very frequent one, and spares no *age.* Nursing infants are particularly apt to be attacked, chiefly on account of sensitiveness of the intestinal mucous membrane in childhood, and the facility with which their food becomes decomposed. Luzsinsky's compilation of ten thousand cases of children's diseases shows, that intestinal catarrh forms almost one-third of the total number of the affections of childhood. At the period of dentition, infants are more subject to enteritis than at other times.

Meteorological conditions are well known to influence the frequency of the disease. The hottest months are regarded as particularly dangerous in this respect, and the number of cases of intestinal inflammations increases especially at the time when there is a rapid alternation of the day and night temperatures.

Sometimes intestinal inflammation assumes an *endemic* or *epidemic* character. This may be explained in some cases by the meteorological conditions above mentioned, or by the use of bad drinking water, and similar general causes affecting large numbers of persons; but in other instances there is probably a miasm at work, the nature of which, however, is entirely unknown (see below under Cholera Morbus).

Symptomatology.

The symptoms of intestinal catarrh are usually not very characteristic, at least they vary considerably in their degree of development in different cases, and are very irregular in their occurrence. Furthermore, the differences in the anatomical structure and the functions of the various divisions of the intestine must modify the symptoms to such a degree, according as the inflammation is differently situated, that it will be impossible to give a general description which is applicable to all cases of enteritis. The best plan, therefore, is to first analyze the symptoms separately, and afterwards to give portraitures of special forms of the disease.

Decidedly the most important and most characteristic symptom of intestinal inflammation is the *diarrhœa.* In the present condition of our knowledge this symptom is chiefly the result of an irritation of the nerves, whereby a more active peristalsis is excited (see above). In catarrh of the intestinal mucous membrane the extremities of the nerves concerned in the reflex production of the peristaltic movements are irritated not only by the inflammatory exudation, but also to a greater degree than usual by the ingesta. This irritation excites contractions of the intestinal wall and a rapid propulsion of the ingesta, which will be discharged externally in a fluid form if the active movements extend also to the large intestine, so as to prevent the proper condensation of the fæces in this portion of the intestinal canal. We know that the chyme, even under normal circumstances, passes through the small intestine very rapidly (within a few hours); it is not of much importance, therefore, as regards the occurrence of diarrhœa, whether the passage of the chyme from the stomach to the cæcum is accomplished somewhat more rapidly than the time mentioned or not, provided the delay in the large intestine is of the normal duration. Hence, when the catarrh is limited to the upper portion of the intestine, particularly the duodenum and jejunum, there may be *no* diarrhœa at all. On the other hand, when the catarrh affects *the lower portion of the ileum and the colon*, diarrhœa occurs, and the more severely the larger the surface of the colon involved in the inflammation. It would certainly be erroneous, however, if we were to conclude that extensive catarrh of the small intestine was wholly without influence in the causation of diarrhœa. When we recall the very important part which the mucous membrane of the small intestine plays in the absorption of ingesta, and the functional derangements which the villi necessarily undergo in enteritis, partly from the inflammatory infiltration of their parenchyma, especially their musculature, and partly from the increased peristalsis which propels the chyme too rapidly over their surface to permit a proper amount of absorption, we can readily understand that in enteritis a large proportion of the food, especially the fats, must pass into the large intestine unabsorbed and half digested. To be sure, absorption, even of fats, may still take place energetically in the large intestine, as I have shown by experiments upon dogs, but this occurs at all events only exceptionally; a part of this excess of fat which passes into the colon there undergoes further decomposition, and gives rise to acids which may in their turn act as irritants to the mucous membrane and excite diarrhœa.

In uncomplicated *catarrh of the rectum*, the evacuations, instead of being thin, are, for obvious reasons, generally consistent. In fact, it is often an accumulation of hardened fæces which induces the proctitis ; while, on the other hand, the spasmodic contractions of the sphincter, excited by the proctitis, tend to produce a still further retention of the fæcal masses.

In *chronic intestinal catarrh*, also, diarrhœa is not a necessary symptom ; on the contrary, there is frequently merely an alternation of fluid and solid stools. This temporary retention of fæces is to be explained, not by an absence of inflammatory exudation, but by the slowness of the intestinal movements. As in chronic catarrh of other mucous membranes, the irritability of the nerve extremities, and the reflex symptoms occasioned thereby, decrease in intensity after a while, so in chronic intestinal catarrh the activity of the peristalsis gradually subsides. This result is favored particularly by the copious muco-purulent exudation which covers the mucous membrane and protects it from the irritation of the ingesta, as well as by the œdematous inflammatory infiltration of the muscularis.

According to the severity of the catarrh, the frequency of the discharges will naturally vary considerably, from four to ten or more daily.

More important than the number is the *character* of the stools. In appearance they vary, according to the intensity of the attack, from thin pulpy fæces to a watery-greenish yellow, or finally an entirely colorless fluid. Their chemical composition has never yet been satisfactorily ascertained.

The very careful analyses by Radziejewski, who administered cathartics to dogs and examined the diarrhœal discharges, show the following ingredients: intestinal ferments (a saccharifying and a peptonizing ferment), leucin, tyrosin, peptones, and occasionally completely undigested food (bundles of muscular fibres), together with traces of unaltered bile—the latter, however, being sometimes entirely absent.

Considerable quantities of chloride of sodium have also been found in diarrhœal stools. But neither this ingredient nor the presence of considerable *albumen* can be regarded as pathognomonic of intestinal catarrh.

Blood is a very rare admixture, except in cases of rectal catarrh, and of the enteritis which results from burns. In the latter affection, however, it is not the hemorrhage which is pathognomonic, but rather the intervention of a latent stage between the reception of the injury and the implication of the intestine, a period which lasts, according to Curling,[1] usually as long as ten days. If, therefore, with the exceptions above indicated, blood be found in the discharges of patients who have for a long time suffered from diarrhœa, we may suspect that we have to deal, not with a simple intestinal catarrh, but with deep structural changes in the intestinal wall, such as ulcers, etc. In cases of doubt, whether the abnormal color of the fæces proceeds from blood or not, the question is best decided by a chemical or a *microscopical examination.* The latter shows—in addition to the red blood corpuscles—unchanged particles of food, *altered epithelial cells*, or *pus corpuscles* (white blood globules), which are not, however, always to be regarded as products of inflammation, because they are found also in the normal enteric juice. No particular significance also can be attached to the presence of *fungi* or *crystals of ammoniaco-magnesian phosphate*, because they occur in connection with the normal putrefactive changes which take place in the large intestine.

The presence of *mucus* in the dejections requires special consideration. Small quantities of mucus are not pathological, but a considerable quantity is met with only in morbid conditions of the intestine. Sometimes the stools consist entirely of mucus, and then a catarrh of the rectum may always be inferred ; so, also, the passage of scybalæ covered with mucus points to an inflammation of the large intestine. In rare cases, particularly in hysterical women, coherent *cylinders of mucus* are discharged in the form of membranous casts of the intestine, from an inch to a foot in length. Their discharge is accompanied by attacks of colicky pains (often above the umbilicus), distention of the abdomen, and an aggravation of the previously existing obstinate dyspepsia (Da Costa). The masses are composed almost entirely of mucin, but sometimes of albumen and fibrin (Whitehead). In some cases these casts are formed in great abundance, and may appear in every stool for months; but usually the attacks last only a few weeks.

The relaxed condition of the skin, increased thirst, concentration of the urine, etc., which are occasional symptoms of enteritis, are to be regarded as the *results* of the excessive or long-continued loss of fluids.

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[1] *Bamberger*, Krankheiten des chylop. Systems. S. 310. cit. Vergl. *Canstatt's* Jahresber. 1870. II. S. 157.

Another symptom of intestinal catarrh, less constant than the diarrhœa, is *abdominal pain*. Its character varies considerably; sometimes it is remittent and colicky, sometimes continuous and dull, and aggravated by strong pressure upon the abdomen; but at other times, as with colic in general, it is rather diminished by pressure. If the inflammation be located in the lower parts of the colon, tenesmus occurs, in its highest degree, if the rectum itself be inflamed. In typhlitis the pain is fixed in the ileo-cæcal region, and becomes so extremely violent, when the inflammation extends from the mucous membrane to the peritoneum, that the abdomen at this point is sensitive to the slightest touch. In the diarrhœa arising from burns the pains are also said to be particularly severe. On the other hand, when the intestinal catarrh has developed gradually without acute symptoms, the pains are less intense—in fact, they may be entirely absent, or they occur only when the peristaltic movements become more active than usual. Generally, however, the patient experiences uncomfortable sensations in the abdomen, particularly after eating; he suffers from borborygmi and flatulence, resulting from the gases generated by the abnormal decomposition of ingesta in the intestines, becomes depressed in spirits, and loses his energy. The escape of the gas by the anus, or by eructation, gives temporary relief by diminishing the distressing tension of the abdomen.

The more severe abdominal pains radiate in various directions, and may even extend to the extremities and genital organs. In young children, convulsions, restlessness, flexion of the thighs upon the abdomen, etc., take place.

In such severe cases the *general health* also suffers severely. Fever is present in acute cases, sometimes ushered in with a chill, and accompanied by headache, loss of appetite, etc. In the catarrh of children the languor, the pallid, cyanotic color of the skin, and the marked general depression, are marked symptoms. If the diarrhœa be long continued, the nutrition inevitably suffers—in fact, even marasmus, with cachectic œdema, may ultimately result from simple chronic intestinal catarrh. The appetite is frequently impaired; and this is the case especially if the inflammation of the mucous membrane be situated near the stomach. Whether in all such cases the stomach is secondarily implicated in the inflammatory process, is very questionable. If, however, we admit that the sensation of hunger is dependent, in part, upon the nerves of the small and large intestines, it is easy to see how vascular and functional derangements of the bowels may of themselves be sufficient to diminish the appetite and even excite nausea.

The *physical examination* unfortunately affords but little aid in the diagnosis of intestinal catarrh. Gurgling and tympanites are, to be sure, met with when there has been a considerable evolution of gas in the intestine, and the musculature has lost its tonicity; but these signs are obviously much too vague and untrustworthy to justify any positive conclusion. In *typhlitis stercoralis*, however, the examination of the abdomen is more satisfactory. In the cæcal region is found a hard tumor, dull on percussion, at first but little sensitive to pressure, but becoming increasingly painful as the inflammation advances. Even after the fæcal accumulation has been removed, a certain amount of swelling is still left, which is due to the inflammatory infiltration, and requires a considerable time for its removal. If *perityphlitis* supervene, the tumor becomes still larger. When this affection occurs primarily—that is to say, independently of any intestinal inflammation—the tumor is characterized by its deep situation, and by its being covered by the cæcum, which, if it be not impacted with fæcal masses, is still tympanitic on percussion. Usually, however, in typhlitis cæcal impaction is present, and then the gases accumulate in the intestine above the ileo-cæcal valve, so as to produce meteorism. A palpable tumor in this region will also be found when a dropsy of the vermiform appendix occurs in the manner described above, from the retention of a considerable amount of secretion.

The existence of an inflammation can, of course, be most readily demonstrated when the disease affects that portion of the intestine which is most accessible to examination, viz., the *rectum*. In this case a digital examination is extremely painful;

the sphincter usually contracts spasmodically, the mucous membrane feels hot and dry, and the finger, when withdrawn, is covered with thin mucus or blood. Inspection with the endoscope, under anæsthesia, will remove all doubt. Chronic catarrh of the rectum is characterized by an abundant discharge of pus, which sometimes oozes constantly from the anus, so that a simple inspection reveals the nature of the case. Not unfrequently the intestinal catarrh is accompanied by *hemorrhoids* and *periproctitis*, which give rise to characteristic, easily recognizable changes in the vicinity of the rectum. When a hard or subsequently fluctuating tumor can be felt projecting against the rigid wall of the rectum and narrowing its lumen, or when a fistulous opening can be detected in the wall of the rectum, periproctitis may be diagnosticated with certainty. The nature of the case is even still more obvious if tenderness be found in the buttock or near the anus, or if perforation have already taken place externally, with the discharge of fetid pus or pus containing fæces. The diagnosis is also clear when the rupture has occurred through the vagina, but is more difficult if the matter be discharged through the bladder or uterus.

Besides the general symptoms of intestinal inflammation above mentioned, there are still others which point to special localizations of the disease, and which will therefore be considered under the individual forms of intestinal inflammation described below. A brief sketch of these forms is necessary for the purpose of aiding the diagnosis of the particular locality affected ; but it should be remarked, beforehand, that in many cases such a differential diagnosis is at present impossible.

Inflammation of the mucous membrane of the duodenum can be diagnosticated only from the presence of *icterus* catarrhalis, all other causes of icterus having been excluded. Generally the duodenal inflammation arises from the *extension of a gastric inflammation* induced by indiscretion in eating. In these cases symptoms of gastric catarrh are present for days or weeks before the conjunctivæ begin to turn yellow. The other symptoms which have been regarded as pathognomonic of duodenal inflammation—severe pain in the right hypochondrium, with vomiting occurring from three to six hours after eating, the passage of fat in the stools, slight nervous symptoms, such as irritability or a sense of terror, especially after eating; or more serious mental disturbances, such as profound sopor, dyspnœa, asthma, etc.—have either proved to be merely symptoms of other affections which happened to coexist with the duodenal inflammation, or else the supposed connection has been only a theoretical inference. Even the icterus itself is not an invariable accompaniment of inflammation of the duodenum; it is occasionally absent in both the acute and the chronic forms of the disease. In many cases, therefore, the existence of the affection may not even be suspected; but the possibility of its presence should always be borne in mind whenever pain, increased by pressure and limited strictly to the right hypochondrium, is developed after an attack of indigestion, exposure to cold, or a burn of the skin. The urine should be examined for the coloring matter of the bile, and we should be on the look-out for the yellow color of the skin which usually makes its appearance somewhat later.

Mayer describes a case in which the inflammation was evidently almost entirely confined to the duodenum (l. c., p. 36): sudden severe pains in the right side, a rigor, vomiting and purging of a thin, green, flocculent fluid, and fever; the "deep-seated pain in the right hypochondrium" is increased by pressure upon the abdomen; ten bloody discharges daily; vomiting; the conjunctivæ begin to turn yellow; the icterus increases very rapidly; delirium; collapse; death. Necroscopy: pylorus and two-thirds of the duodenal mucous membrane very much inflamed; the orifice of the ductus choledochus was closed by the swelling, so as to be discovered with difficulty. A portion of the transverse colon, although to a far less extent, was also reddened by inflammation.

Catarrh limited to the jejunum or ileum affords still fewer points for diagnosis. As has been already mentioned, intestinal catarrh in this locality does not necessarily result in the production of diarrhœa. To take the diarrhœa of typhoid fever, with its accompanying symptoms, as the prototype of ileitis in general, would be to ignore the fact that diarrhœa may be entirely absent in enteric fever, and that, on the other hand, after a long-continued diarrhœa in typhoid fever, the intestinal mucous membrane, after death, sometimes appears pale, with scarcely any indications of ulceration;[1] these facts proving that in typhoid fever the diarrhœa should be regarded as due chiefly to other causes than the complicating ileitis—probably to nervous influences. Inflammation limited to the small intestine may be *suspected*, however, if, in addition to symptoms of gastritis, colic, marked rumbling in the bowels, and a feeling of active propulsion of the ingesta, are observed, without the occurrence of diarrhœa. When diarrhœa does occur as a result of extension of the peristalsis to the colon, or of an implication of this part in the inflammation, a catarrh of the small intestine may be diagnosticated, if the dejections contain large quantities of the ingredients and products of the secretions of the duodenum and small intestine, viz., unaltered bile, intestinal ferments (the saccharifying and peptonizing ferments), leucin and tyrosin in distinctly appreciable quantities, a considerable amount of entirely undigested food, and, particularly, much undigested fat.

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[1] See, further on, a case of this kind in the chapter on Intestinal Ulcers.

Colitis, which is the most common form of intestinal catarrh, manifests itself by colicky or more continuous localized pains, and diarrhœa, with discharge of mucus, pus, and perhaps also the above-described cylinders of mucus, which are probably formed in the large intestine. In *chronic* cases, in which the symptoms correspond, upon the whole, to those of chronic intestinal catarrh, as generally described, there is usually an alternation of constipation with the diarrhœa; besides which, the patient suffers also from meteorism, flatulence, dyspnœa, in consequence of the impeded movements of the diaphragm; troublesome palpitation, derangements of nutrition, resulting from lack of absorptive power on the part of the intestines; uncomfortable feelings in the abdomen, nausea, eructations, and sometimes even vomiting. The vomiting is probably generally due to a coexisting gastritis, particularly in those cases in which the entire mucous membrane of the digestive tract is in a condition of passive hyperæmia and chronic inflammation, resulting from obstructed circulation, as in emphysema, cirrhosis of the liver, etc. Finally, these patients suffer also from very marked depression of spirits, or from true *hypochondria;* but how far such symptoms are to be ascribed to a chronic poisoning of the central nervous system by the absorption of abnormal products of digestion, it is as yet impossible to decide. Upon this point the reader is referred to my remarks upon chronic gastric catarrh.

The picture presented by *inflammations* of *the cæcum and vermiform appendix* is quite characteristic. It is true that *typhlitis* runs its course with a great diversity of symptoms, some of a mild and others of a severe character, but usually the symptoms are sufficiently typical to render the diagnosis an easy one. We shall confine ourselves at present to the description of the simple form of typhlitis (stercoralis), which usually terminates in recovery ; the severe form, ending in deep ulceration and perforation, will be considered in the chapter on Ulcers of the Intestines. The symptoms at the outset are of a vague and very insignificant character, such as dyspepsia, constipation, and distention of the abdomen. Soon a distinct tumor, which is generally tender on pressure, makes its appearance in the cæcal region, the patient complains of pain in the right thigh, the constipation becomes more marked, gases accumulate above the obstructed point, and vomiting ensues, which ultimately becomes of stercoraceous character. The patient now presents the aspect of a most serious disease, and death may take place with symptoms of collapse even without the occurrence of perforation. A change for the better is, however, not impossible even when the symptoms are most severe. In this event the bowels begin to move, and large quantities of fæces are discharged, the severe pains disappear, and the general condition of the patient improves ; but even after the complete evacuation of the cæcum, a tumor very commonly still remains as a result of the inflammatory infiltration, the movements of the bowels are irregular, and a chronic typhlitis is very apt to ensue after the subsidence of the acute symptoms.

In those cases of *perityphlitis* in which the inflammation of the cellular tissue around the cæcum is the chief lesion or occurs as the primary affection, there is *less meteorism*, while a feeling of numbness and formication is experienced in the right limb, with sharp pains on movement of the same. The tumor in the cæcal region is more deeply seated, and at the outset may be unaccompanied by any symptoms of intestinal catarrh, but later in the attack constipation and nausea supervene. The disease may terminate in various ways: by absorption of the exudation, by the formation of an abscess which bursts externally at a more dependent point without implicating the cæcum, or bursts through the cæcum with escape of the pus by the stool—all of which results are favorable—or, finally, and almost always fatally, by a rupture of the abscess into the peritoneal cavity, or by the occurrence of pyæmia and collapse.

When the inflammation is confined to the *vermiform appendix*, it will manifestly escape recognition, unless it be accompanied by other symptoms, such as the above-described dropsical enlargement of the canal, or the occurrence of perforation resulting in a peritonitis which begins in the right inguinal region.

In *proctitis* the characteristic symptom is the presence of severe tenesmus, a feeling as if something were sticking in the rectum and had to be forced out. The sphincter ani becomes spasmodically contracted, the discharge of urine is irregular and accompanied by strangury, and the pains become most intense when hard fæces covered with blood and mucus are discharged. In the milder cases the patient complains merely of a burning sensation in the anus, with a painful and unsuccessful inclination to evacuate the bowel. At short intervals he tries to obtain relief in this way, but on almost every attempt succeeds in discharging only a small quantity of bloody mucus, while in some cases the wall of the rectum is forced down in the form of a dark-red cushion of mucous membrane. The disease may end in recovery, or it shows a tendency to become chronic, in which case the pains grow less severe and assume more of a tensive or dragging character. The secretion covering the rectal mucous membrane now becomes purulent or sero-purulent, and the simple catarrh changes to a chronic blennorrhœa. Pus, either in a pure form or mixed with the stools, is discharged from time to time with tenesmus, or it sometimes oozes from the anus continually. The stools vary in character, being sometimes constipated, sometimes loose, and sometimes mixed with blood from the swollen hemorrhoidal veins. In both its acute and chronic forms the inflammation may extend to the cellular tissue about the rectum. In this case the finger introduced into the rectum detects induration of the wall, or in the later stages fluctuation and narrowing of the canal by the tumor. When the swelling is situated in the parts surrounding the anus, we have the usual symptoms of an abscess tending to rupture. By the discharge of the pus externally, into the rectum or into some adjacent cavity, rectal fistulæ are formed, the "external," the "internal," the "complete," or the recto-vaginal, recto-vesical, etc. These results may produce dangerous complications, such as infiltration of urine and pyæmia. Instead of terminating in abscess, however, the periproctitic exudation is occasionally absorbed, and then leaves after it a chronic induration of portions of the wall of the rectum. The *idiopathic periproctitis*, which is said to develop with special frequency in tuberculous patients,[1] runs the same course as the secondary form, except that it does not begin with marked inflammatory symptoms in the rectum ; on the contrary, the patient at first complains only of constipation with pain in the rectum, especially on movement of the bowels, until after a while unmistakable signs of suppuration, etc., supervene. In rare instances symptoms of ileus may develop during the course of the periproctitis, as was shown in a case observed by Boens.

In the consideration of cholera-morbus on page 147, we have already given a brief description of the form of this affection, which occurs in infancy, and which is characterized by vomiting and purging, rapidly followed by collapse. A more complete description of the enteritis infantum in its strict sense would be out of place in this article, and we shall, therefore, confine ourselves to a brief mention of the more chronic or intermitting forms of diarrhœa, which during the early years of life so frequently interrupt the normally quiet development of the organism at this period. The question whether all cases of diarrhœa are due to an *inflammation* of the intestine has already been considered in the beginning of this chapter. The absence of any intestinal lesion at the autopsy, as has been observed occasionally in cases of infantile entero-colitis (see also Bouchut, l. c., p. 753–54), is of course no positive proof that inflammation did not exist during life, because, as already mentioned, all signs of hyperæmia of the mucous membrane may disappear after death. Still, I think one needs only to have observed for himself how a temporary cold will excite a slight diarrhœa without any constitutional disturbance whatever, to become satisfied, also, that a purely nervous derangement without inflammation may occasion a looseness of the bowels merely by increasing the peristaltic movements of the intestines. Such influences must play a part also in the diarrhœas which occur in young children at the period of dentition. The influence of teething in the causation of infantile diarrhœa may be still an open question, but that the association between the two conditions is certainly a very common one is recognized by physicians of the most experience in children's diseases no less generally than it is implicitly believed in by mothers. In many cases the diarrhœa comes and goes with the cutting of each tooth. At other times the enteritis and diarrhœa are preceded by other causal conditions, such as the influence of cold, derangements of nutrition, the unavoidable change in diet on weaning, helminthiasis, or some one of the above-mentioned causes which are specially apt to excite intestinal catarrh in adults. Usually the attack of diarrhœa is ushered in by restlessness, cries indicative of pain, and even convulsions. The frequent fluid dejections, which at first are of normal appearance, soon become of a grumous consistency resembling minced eggs, and greenish-yellow in color. They are generally of an acid odor and reaction, and contain, besides the fæces and entirely undigested food (lientery), a certain amount of lumpy mucus. The intensely acid discharges very often redden the parts around the anus. Meteorism, resulting from the imperfect resistance presented by the relaxed intestinal wall to the gases evolved from the ingesta, accompanies both the acute and the chronic form of intestinal catarrh, but particularly the latter, so that the abdomen is tympanitically distended ("frog's belly"), while the round figure of the child at other parts of the body has disappeared, the skin is pale, the face appears wrinkled and old, the eyes sunken, and the angles of the mouth and alæ nasi wear a marked expression of pain. The skin becomes cool, while, on the other hand, the abdomen is occasionally excessively hot; the movements are languid, the sphincter is paralyzed, and the mouth covered with aphthæ. The appetite is not necessarily lost, nor is vomiting invariably present, but there is increased thirst, due to the great loss of water, especially in the acute form of the disease. In some cases there is but little fever; but the course of the temperature has not been closely investigated. When the intestinal catarrh is of long duration, death is the usual result, especially if ulceration of the follicles have supervened. The chronic intestinal catarrh of children is also accompanied by an enlargement and fatty condition of the liver, analogous to the similar lesions in tuberculous patients, or the case may result in amyloid degeneration of the abdominal organs, in rachitis, or in scrofula—the scrofulous disease beginning first in the mesenteric glands, which become so much enlarged as to be palpable through the abdominal wall.

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[1] Among the very numerous cases of phthisis which have come under my observation within the past six years, I have never met with an instance of "tuberculous" periproctitis.

Diagnosis.

The *diagnosis* of inflammation of the intestines is, as a rule, not difficult, when attention is paid to the symptoms above mentioned. The precise determination of the locality of the inflammation is, however, not only difficult, but in many cases impossible, as must be sufficiently evident from the previous description of the individual symptoms of the disease. If the case be one of simple catarrh, the question to be determined, so far as the treatment is concerned, is *whether the inflammation affects the large or the small intestine.* The decision, as above mentioned, will rest chiefly upon the history of the case and the character of the stools. *Inflammation of the small intestine* is indicated by the accession of the intestinal catarrh after a well-marked gastritis, by frequent vomiting, icterus, the detection of unaltered products, and ingredients of the secretions of the duodenum and small intestine in the dejections, the appearance of undigested food shortly after it has been taken, and by a marked derangement of nutrition. On the other hand, tenesmus, the admixture of undecomposed blood in the dejections, the envelop-ment of the fæcal masses in mucus, the discharge of pure mucus or pus, and the sensitiveness of certain points in the course of the colon, are signs which directly indicate *inflammation of the large intestine.*

In *typhlitis* and perityphlitis the diagnosis presents less difficulty, at least so far as the location of the disease in this neighborhood is concerned. The latter affection may be diagnosticated, in distinction from typhlitis, by the sharp pains and anomalous sensations in the right leg, and by the slight degree of *meteorism*, in contrast with the greater accumulation of gases which takes place when an obstruction is occasioned in the intestine by a typhlitis. Perityphlitis is also to be distinguished by the deep situation of the tumor in the cæcal region and by the occurrence of burrowing abscesses. *Whether the vermiform appendix or the cæcum forms the special starting-point for the inflammation*, it is usually impossible to decide, especially when a copious exudation has taken place. But, since experience shows[1] that cases terminating in peritonitis originate far more frequently in the vermiform appendix than in the cæcum, the chances in doubtful cases will be in favor of an inflammation of the appendix. Finally, the recognition of *catarrh of the rectum* by the complex of symptoms above given presents no difficulties.

And yet, both in inflammation of the rectum and in inflammation of other parts of the intestines, it is not always easy, on the one hand, to distinguish with certainty between an idiopathic and a secondary catarrh—in other words, to diagnosticate the fundamental disease which is running its course with the symptoms of a simple catarrh—and, on the other hand, to exclude at once the morbid conditions which induce symptoms resembling those of intestinal catarrh. It will be necessary, therefore, to devote a few words more to the *differential diagnosis.*

An acute or chronic catarrh, extending over a large surface of the small and large intestines, cannot easily be mistaken for any other affection, except, perhaps, typhoid fever; but at the present day, when the great importance of the thermometer as a guide in the diagnosis of the latter disease is generally recognized, this error is less to be feared than formerly. In doubtful cases the course of the temperature, the enlargement of the spleen, the roseola, bronchitis, etc. (see above, Gastric Catarrh), will decide the question.

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[1] Bamberger, out of eighteen fatal cases of peritonitis starting in the right iliac region, found that in fourteen the cause was ulceration of the vermiform appendix, and in only three or four inflammation of the cæcum (l. c., S. 340).

Typhlitis, with fæcal impaction of the cæcum, presents, when peritonitis also exists, so striking a picture that the diagnosis is comparatively easy. And yet when the typhlitic tumor undergoes a *gradual* development, it is liable to be confounded with the whole class of abdominal tumors located below the umbilicus, such as renal, ovarian, and retroperitoneal tumors. All of these tumors, as contrasted with the typhlitic, have, however, this peculiarity, that they are covered by convolutions of intestine, particularly by the tympanitically resonant ascending colon laterally. Even when the colon is filled with fæcal masses, the nodular scybalæ can generally be distinguished from the massive tumor lying underneath, to say nothing of the evidence to be derived from examination of the urine, palpation by the vagina, and the history of the case.

Occasionally great difficulty may arise in determining whether the intestinal obstruction is produced by carcinoma of the cæcum and ascending colon, by intussusception of the ileum into the cæcum, or by typhlitis stercoralis. A very gradual development and nodular hardness of the tumor, together with a cachectic condition of the patient, point to carcinoma of the cæcum; while if the tumor be elastic on palpation, of a longitudinal, sausage shape, and absolutely dull on percussion, if it move forwards along the course of the colon, and if it can be ascertained that the formation of the tumor has been preceded by persistent diarrhœa, the diagnosis of intussusception is in a high degree probable, and becomes certain if the invaginated portion of intestine can finally be felt by the rectum or after a few weeks is discharged in a gangrenous condition by stool.

The diagnosis of *proctitis* is obviously almost always very easy, and usually the only difficulty is to determine, not whether inflammation of the rectum is present, but to what cause it is due

—in other words, whether the catarrh is of a simple character, or is complicated with some serious fundamental disease, such as carcinoma, ulcers, or hemorrhoids. In such cases the manual examination of the rectum and the inspection by means of the endoscope or speculum will be the most important guides. From infective dysentery proctitis is not to be distinguished, so long as there is no discharge of the reddish yellow, flocculent fluid, which is characteristic of the former affection, nor any marked constitutional disturbance.

Prognosis.

In inflammation of the intestines, the prognosis varies considerably, according to the severity of the disease, the cause which produces or maintains it, the constitution of the patient, etc., and the following remarks will refer therefore only to general points of leading importance.

While in adults an idiopathic catarrh extending over a large extent of intestine is devoid of danger and usually terminates in recovery sooner or later, it is justly regarded as an affection much to be apprehended in children, and the more so the younger the child. So, also, a simple catarrh in advanced life is dangerous, and may lead to a fatal result from exhaustion, since the derangements in the transformation and absorption of the food, conditioned by the lesions in the intestinal mucous membrane, react, when long continued, upon the entire organism. So striking is the impairment of nutrition in the intestinal catarrh of children, that this variety of the disease has been designated "atrophia infantum." Even if death do not occur during the attack, it is very likely to ensue subsequently, as a result of the sequelæ—scrofula, amyloid degeneration, etc. Chronic intestinal catarrh may lead also to a *simple stricture of the intestine*, which, even after the original inflammation has subsided, may induce chronic retention of fæces, and thereby occasion a renewal of the intestinal inflammation. Among the acute inflammations, those which occur after burns are regarded as specially dangerous, because they are apt to be followed by hemorrhage, ulceration, and fatal collapse.[1] Nor can much improvement be hoped for in most of the cases of *secondary* catarrh. Since the disease is here only the result or symptom of some other affection, the prognosis depends of course entirely upon the severity of the latter, and it need scarcely be mentioned that intestinal catarrh, occurring in the course of septicæmia or uræmia, is relatively more dangerous than when it is a complication of emphysema or some similar protracted disease attended by chronic stasis.

By far the most dangerous locality for the occurrence of enteritis is the cæcum, as must be apparent from the serious complications which arise, as we have seen, during the course of *typhlitis*. Recovery is, however, the usual result in typhlitis stercoralis, in idiopathic perityphlitis, and even in inflammation of the appendix, provided no perforation or extensive peritonitis have occurred. Bamberger's compilation of seventy-three cases observed by himself, in all of which considerable exudation was found in the right iliac region, shows a mortality of only twenty-five per cent. But even when the attack does not prove fatal, it is apt to give rise to sequelæ, which seriously affect the prognosis, such as stenosis of the intestine, displacements, adhesions to the surrounding parts, involving the danger of subsequent incarceration, burrowing abscesses with caries of bone, fæcal fistulæ, pylephlebitis—the results of which often manifest themselves only after a considerable time—and especially, also, a tendency to relapses.

Proctitis, finally, is attended with danger only when its causes are irremovable. Thus all the inflammations of the rectum which are due to the presence of foreign bodies, fæcal masses, worms, etc., in the rectum, present a favorable prognosis in contrast with the proctitis which accompanies tuberculous ulcers, cancer of the rectum, pelvic tumors, or passive congestions of the hemorrhoidal branches of the vena portæ. If symptoms of *peri-*

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[1] In a case of burn of the skin reported by Sutton (Canstatt's Jahresber. 1870), diarrhœa set in on the day following the burn—the patient having in the interval felt perfectly well—and death ensued three hours afterwards. At the autopsy the only lesion was injection at various points on the intestinal mucous membrane, a striking contrast to the serious symptoms which the pulseless, livid, collapsed patient had presented during life.

proctitis have supervened and have resulted in rectal abscesses and fistulæ, the prognosis is less favorable, and still more so if the patient be in poor health or in fact tuberculous. Proctitis, produced by rheumatic influences (catching cold at stool, etc.) or by local injury, runs almost invariably a favorable course, as does also the periproctitis excited by similar causes.

Treatment.

In all cases of intestinal catarrh, whether recent or chronic, whether situated in the upper or in the lower portions of the intestines, *the selection of a suitable diet* should be regarded as probably the most important part of the treatment. Aside from the loss of power on the part of the inflamed intestine to properly transform and absorb the food, it should never be forgotten that the chemical and mechanical irritations which the chyme and the more solid fæces normally excite in the healthy mucous membrane may act abnormally when the mucous membrane is inflamed. All food, therefore, which is difficult of digestion, and the mechanically irritating portions of which of course pass into the large intestine still undigested, such as leguminous vegetables, cheese in large pieces, cabbage, stringy meat, etc., must be strictly avoided ; so, also, but little bread should be allowed, because, as Voit's experiments have shown, a bread diet increases the solidity of the fæces. In general the best diet for patients with intestinal catarrh will consist of albuminous substances of an easily divisible character, such as milk, yolk of egg, meat solution, food and broths containing but little starch, and especially the various kinds of mucilaginous substances, such as barley-water, groats, etc. In addition to the indigestible articles above mentioned, fatty food is also to be forbidden, because, in consequence of the increased peristalsis and the unquestionably diminished power of absorption on the part of the intestinal villi, most of the fat passes undigested into the colon, and there undergoes decomposition. As to the *details* of the comparative digestibility of different articles of food, I refer to the chapter on Chronic Gastric Catarrh. When the disease occurs in young infants, the selection of the diet requires special care, because here we have to deal with a mucous membrane which, even in health, is extremely sensitive and generally tolerates but *a single* variety of food. In many of these cases the best prescription is a wet-nurse. In other respects the regimen suggested in the treatment of cholera infantum (see above) will apply also to the more chronic intestinal catarrhs of children.

Next to the proper regulation of the diet, *one of the most important measures for the rational treatment of inflammation of the intestines is the removal of irritating ingesta, and particularly any decomposing fæcal masses that may happen to be present in the intestinal canal.*

This precaution should never be neglected, even when the presence of diarrhœa seems to contraindicate the administration of cathartics. If the patient has eaten coarse indigestible food before the beginning of the diarrhœa, or at least before he has come under treatment, if small scybalæ are found in the fluid stools, or the colon is dull on percussion at particular points, or, finally, if the attack has been preceded by constipation, it is good practice to begin the treatment by administering a clyster or some mild cathartic like castor-oil. Indeed, even a thorough washing out of the intestine is necessary if the inflammation is located in the lower portions of the large intestine, or if threatening symptoms arise in consequence of an obstinate fæcal impaction in some portion of the intestine behind the ileo-cæcal valve.

Another general rule in the treatment of intestinal catarrh is, to confine the patient to his bed in every case that is not entirely unimportant. Neither snugly-fitting undergarments nor a flannel abdominal bandage can take the place of the uniform warmth of the bed, which is all the more indicated because this disease is undoubtedly often dependent upon rheumatic influences. For the same reason, persons who are predisposed to intestinal catarrh should wear woollen stockings, flannel drawers, an abdominal bandage, etc., for the sake of prophylaxis, especially when travelling.

Like the prognosis, the treatment also is determined by the exciting cause of the intestinal catarrh.

The *evacuating treatment*, which has already been mentioned as appropriate in many cases, is necessary, particularly when the inflammation has been excited by indigestible, irritating food, by an accumulation of worms or solid fæcal masses, by foreign bodies and poisons, or by a stenosis of the intestine. If the removal of the irritating substances can be effected by enemata or irrigations, these are to be preferred, on account of their milder action, to the use of purgatives. Still there is no objection to the administration of a laxative after the irrigation, in order to be sure that the intestine is relieved of its irritating contents. The most harmless cathartic for this purpose is the castor-oil emulsion (ol. ricini f. ℥ j. pulv., acaciæ ℈iv., syr. f. ʒ vj., aquæ dest. f. ℥ v. M. S. one tablespoonful every two hours until it operates). In place of the oil, calomel may be given, which acts promptly, empties the upper portions of the intestines (Radziejewski), and does not appear to aggravate the intestinal inflammation. The *only* form of intestinal inflammation in which the use of cathartics is attended with much risk is *typhlitis*, and this objection applies equally to the stercoral form of the disease, notwithstanding the apparent indication for their employment. When the peritoneum in the neighborhood of the cancer has become involved in the inflammatory process, a cathartic, by exciting a more active peristalsis, may tear the salutary adhesions which have already formed, or may even produce a rupture of the cæcum at a point which has been thinned by ulceration. Only, therefore, at the very outset of typhlitis, or while the cæcal region is but little sensitive to pressure, are cathartics allowable, and then only after injections have been persistently used without a satisfactory result.

On the other hand, cathartics may be prescribed with advantage when the intestinal affection is due to septic or infective processes, such as occur in septicæmia and uræmia, in the latter of which drastics have long been the customary treatment.

It is interesting to observe how our present practice of beginning the treatment of diarrhœa with the administration of a cathartic, or at least of not arresting the discharges at the outset of the attack, was also recommended by the older writers. Thus, Celsus (l. c.): "Primo die quiescere satis est; neque impetum ventris prohibere." And Pemberton says: "The treatment should usually begin with a cathartic."

When the diarrhœa is the result of *malarial infection*, the treatment should begin with large doses of quinine—a remedy which experience has shown can be given in the obstinate diarrhœas, which occur in persons living in malarial regions, without exerting any injurious influence upon the intestinal canal.

In all intestinal inflammations induced by *exposure to cold*, it is important to secure *diaphoresis*. The good effects of sweating, in ordinary colds, has always been recognized ; but aside from the hope of counteracting, in this gross allopathic way, the harm done to the intestine by the chilling of the skin, the employment of diaphoresis is especially indicated, also, by the fact that there is a connection between the amount of perspiration excreted by the skin and the amount of fluids discharged by the intestines. Thus in many persons a temporary constipation always excites increased perspiration, and *vice versa*. The best diaphoretics that can be used are vapor or hot-water baths, followed by packing.

When the chronic intestinal catarrh is occasioned by *portal congestion*, arising either from disease of the liver, or indirectly from disease of the lungs and heart, the treatment should usually be addressed rather to the fundamental disease than to the catarrh directly, and remedies like digitalis may be used with the view of relieving the congestion by increasing the force of the heart's action. When the patient has previously experienced relief from the spontaneous occurrence of hemorrhages from the anus, this curative effort of nature may be imitated in severe cases by the application of leeches to the anus, in order to deplete the portal vessels, provided, of course, the patient can bear the abstraction of blood, and does not, on the contrary, urgently require a *restorative regimen*.

Such a regimen is manifestly the chief indication in all those cases in which the occurrence of intestinal catarrh is favored by general debility, or, to speak more correctly, by a weakness of the walls of the vessels, as in tuberculosis, morbus Brightii, etc.

Finally, mention should be made also of the purely symptomatic catarrhs, which result from ulcerations, neoplasms, intussusceptions of the intestine, etc. In such cases the treatment coincides with that of the causal morbid conditions.

Indicatio morbi: As soon as we are satisfied that the irritating ingesta have been removed from the intestine, by the means mentioned above, or that further attempts in this direction would be useless, *the most important indication is to secure rest to the inflamed organ.* This result, as experience has shown in thousands of cases, can be best obtained by opium. No other remedy quiets the diarrhœa so promptly and durably, although we still know but little in regard to the manner in which it influences the peristaltic movements. The average effective dose is half a grain or twelve drops of the tincture, which may be given several times daily. At the same time, the patient should be placed upon a restricted diet, such as barley-water with a little salt, or groats, and a moderate quantity of red wine may be allowed. Gradually the easily digestible diet above mentioned may be resorted to. The strictness with which this regimen is to be enforced will naturally depend upon the severity of the case. If the symptoms of intestinal inflammation still remain uncontrolled, astringents may now be used, unless there are urgent reasons, as in typhlitis, for preventing any further accumulation of fæces. Experience has shown that the "astringing, contracting" action of these remedies exerts a beneficial influence upon the mucous membrane where they come into direct contact with it. The astringents most commonly used in intestinal catarrh are the *acetate of lead* (half a grain per dose), the *nitrate of silver* (from one-sixth to one-third of a grain), *tannin* (from two to six grains, frequently prescribed also in combination with opium[1]), *alum* (from five to eight grains, also given with opium[2]), *sulphate of iron* (from one to

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[1] ℞

Acidi tannici	℈ij.
Tinct. opii	♏ xv.
Syrupi	℥ j.
Aq. menth. pip	f. ℥ v.

M. S. One tablespoonful every two hours.

[2] ℞

Aluminis	gr. viij.
Opii	gr. ss.
Pulv. acaciæ	gr. viij.

M. Ft. chart. tal. No. X.
S. One powder three times daily.

three grains), or the *solution of chloride of iron*[1] (from five to eight drops). Remedies, which are similar in their action to tannin, such as *catechu* (from eight grains to a scruple), and *kino* (from eight to fifteen grains), are also frequently prescribed in chronic intestinal catarrh. A favorite remedy when the stomach is easily disturbed is columbo, given in infusion, which acts also as a bitter, and sometimes gives excellent results. All of these remedies, except the three last mentioned, may be given also by enema,[2] and this mode of administration will obviously be more efficient when the catarrh affects the lower portion of the large intestine. In mild cases the regular use of injections of cold water is often sufficient to produce a slight antiphlogistic astringent effect upon the inflamed mucous membrane of the rectum.

As in many cases of chronic gastric catarrh, so also in catarrh of the intestines, a *course of mineral waters* will often be the most certain means for permanently restoring the diseased mucous membrane to its normal condition. When it is desirable to secure a regular evacuation of the contents of the bowels, the alkaline waters may be used with benefit, particularly the alkaline-saline waters, such as the Tarasp, Carlsbad, Rohitsch, etc., which have enjoyed great reputation for the relief of portal congestions, fatty liver, etc. If the intestinal catarrh is accompanied by marked *nervous irritability* of the patient, as is not uncommonly the case when the disease has been of long duration, it may be well, in addition to the alkaline waters, to try the sedative action of one of the cooler "acratothermæ,[3]" or the patient may be sent to the *mountains*, or *sea-bathing* may be recommended in the milder cases. If hypochondria with constipation be a prominent feature of the disease, one of the chloride of sodium mineral waters, particularly the Kissengen, should be used. The favorable action of this water in such cases is to be explained partly by the fact that the water facilitates the transformation of the food and stimulates the powers of digestion, and partly by its cathartic action when given in large doses. Kissingen water is well adapted, also, to relieve the intestinal catarrh which is attended by diarrhœa. When taken in small quantities, it has rather a constipating than a cathartic effect, and it promotes the gastric digestion, so that the chyme, as it enters the intestines, is in a condition more favorable to its absorption (Diruf). Finally, much benefit may sometimes be derived from the *cold water treatment.* Caliburces has shown that the cold sitz-baths, wet packs around the body, and "slapping with wet towels," all excite peristaltic movements of the intestines. Moreover, the long-continued hyperæmia of the skin, which follows the application of the wet pack, may produce a derivative action from the intestines, similar to that which follows the occurrence of hemorrhoidal bleedings. Recently the use of cold, wet compresses had been recommended also in acute intestinal catarrh, particularly in the diarrhœa of children (Winternitz, Oppenheimer).

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[1] The *stronger* solution of the British Pharmacopœia.

[2] The dose by enema is about twice or three times as large as the quantity used by the mouth.

[3] Thermal springs with no active properties (ἀκρατής).—TRANS.

Strychnine and *belladonna* have been strongly recommended, by some writers, in cases of intestinal catarrh: belladonna for chronic catarrh with diarrhœa and atony of the intestines, and strychnine for chronic constipation in general. My own experience with these remedies for the conditions referred to has been insufficient to enable me to point out any precise indications for their use—in fact, I regard it as improper to employ, or rather to experiment, with remedies of such active properties before we clearly understand their physiological mode of action; and certainly, in regard to strychnine, we possess no information of this kind whatever.

The measures required by the *indicatio symptomatica* have for the most part been already considered, at least so far as the special treatment of the *diarrhœa* is concerned, and also the treatment of the *colicky pains*, which disappear along with the diarrhœa under the influence of opium. This remedy, in the case of rectal catarrh, may be advantageously used in the form of suppositories or enemata. When the colicky pains are occasioned chiefly by an excessive distention of the intestine, carminatives, such as caraway, peppermint, etc., may be given. In severe meteorism, powdered charcoal (fifteen grains at a dose) may also be tried, or the attempt may be made to withdraw the gas by means of an intestine-tube with a central orifice at its lower extremity, or, perhaps still better, by a stomach-pump with a tightly-fitting piston. When *vomiting* occurs, as a complication, opium, morphine injections, ice-pills, etc., are required; while in *collapse*, a condition which assumes special prominence in children, wine and other stimulants are indispensable. There still remain to be considered briefly the indications presented by the locality and specific course of the several forms of inflammation as they affect different portions of the intestines.

In distinction from other forms of intestinal inflammation, *typhlitis*, on account of its marked inflammatory character, requires an energetic antiphlogistic treatment. This is to be carried out by means of the continuous application of frozen compresses,[1] or ice bags, by leeching (in robust individuals), and by injections of ice-water into the large intestine, the latter remedy also serving the purpose of evacuating the bowel, which is always the first and most important indication, so long as no symptoms of peritonitis are present. If the ice-water injections are unsuccessful, the common practice is to add salt or honey to the injection, and at the same time to give a laxative, such as castor-oil, or even stronger purgatives, if peritoneal irritation can be certainly excluded. In desperate cases quicksilver may be given, six ounces at a dose.[2] If these measures do not succeed, and indications of peritonitis supervene, opium should be used, or, as a last resort, enterotomy may be performed at a point above the obstruction, the situation of the latter being determined by the dulness on percussion. The indurated infiltration-tumor remaining after the cessation of the typhlitis is to be treated by poultices, warm baths, or salt-water baths, for the purpose of producing absorption; the perityphlitic and burrowing abscesses are to be opened as soon as fluctuation is detected. Abscesses occurring during the course of *idiopathic perityphlitis* also require an early opening, and, in other respects, are to be treated in the same way as other deeply-situated inflammations, viz., by rest, counter-irritants to the skin, etc. At the same time the bowels should be kept open, in order to prevent pressure upon the inflamed part.

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[1] These can be readily made by placing the wet compress on a table, underneath a shallow tin-pan filled with cracked ice and salt.—TRANS.

[2] Traube has shown that the quicksilver probably acts not so much by its weight as by the strong peristalsis which it excites, beginning in the stomach. The increased peristalsis, of course, involves the danger of a rupture at the inflamed part.

The same principles will apply also to the treatment of *periproctitis*—rest, cold, and afterwards poultices, a regular evacuation of the bowels, injections of warm infusions of chamomile flowers into the rectum, and the opening of fluctuation points, in order to prevent perforation into the bladder or rectum. The treatment of the rectal fistulæ is to be conducted in accordance with the rules laid down in the text-books on surgery.

In *proctitis* the treatment is essentially of a local character, because here we can apply remedies directly to the diseased mucous membrane, or at least in its immediate vicinity, in the form of cold, warm, or astringent enemata, sitz-baths, suppositories, and abstractions of blood from the anus. For severe tenesmus, ointments of extract of opium or belladonna, or narcotic suppositories, may be used, while fissures of the anus may require a division of the sphincter. In cases of prolapse of the rectum occasioned by the extrusion of the hypertrophied folds of mucous membrane, the prolapsed portion should be replaced, touched with nitrate of silver, partially or completely excised, or removed by the actual cautery. The accumulation of fæces should be guarded against in this as well as in other forms of intestinal catarrh.

In regard, finally, to the treatment of the intestinal catarrh of children, I refer to the general principles laid down above, and particularly to the therapeutic measures more fully described in the article on Gastro-enteritis Infantum.

Phlegmonous and Diphtheritic Inflammation of the Intestines.

The intestinal pathological changes in question will be considered very briefly, because they rarely occur as independent affections, but rather, in most instances, as symptoms of other diseases, and therefore possess less clinical than pathologico-anatomical interest.

Enteritis phlegmonosa (submucosa purulenta) corresponds in its anatomical characters to the better known gastritis phlegmonosa, which has already been considered at length. Its occurrence as an independent affection is extraordinarily rare; generally it occurs as a so-called metastatic inflammation, and is most frequently situated in the duodenum. It is occasionally found as a result of intense irritations of the intestinal wall in connection with ulcers. Like phlegmonous gastritis, phlegmonous enteritis may also lead to sclerosis, with contraction of the wall of the organ, or to suppuration and the formation of abscesses, which perforate the mucous membrane or excite other abscesses in the retro-peritoneal connective tissue.

Much more frequent is the *enteritis diphtheritica.* Besides the specific infective diphtheria of the intestine—*dysentery*—and the diphtheritic disease of the bowels which sometimes occurs during the course of contagious *throat-diphtheria* (*vid.* Vol. I.), the mucous membrane will sometimes also be found to be covered with a firmly-adherent, flaky, white or whitish gray deposit. Beginning in the form of scattered islands, the diphtheritic infiltration gradually extends laterally, in the small intestine along the folds of Kerkringius, and in the large intestine upon the ileo-cæcal valve and along the bands and plicæ sigmoideæ.

The severe disturbance of nutrition produced by the diphtheritic exudation leads to superficial necrosis and ulceration, resulting in perforation or in cicatrization, and occasionally, but rarely, in narrowing of the lumen of the intestine. In most cases the diphtheritic inflammation is located in the large intestine, sometimes also in the small intestine, and only in very rare instances is it wholly confined to the latter location. Thus, in the one hundred and three cases of intestinal diphtheria collected by Weissenfels[1] and Freund[2] from the post-mortem records of the Berlin Pathologico-anatomical Institute, the diphtheritic inflammation was confined to the small intestine in only four.

This anatomical fact naturally suggests the inference that the formation of the diphtheritic deposit is due to local mechanical causes, particularly to the action of irritating ingesta at those portions of the mucous membrane where the most friction occurs and the fæces are longest delayed. Thus, the English writers, and among the Germans especially Virchow,[1] long ago pointed out the very important part which retention of fæces in the large intestine plays in the causation of dysentery. When fæcal masses remain impacted for a very long time in portions of the intestine where a stagnation is most liable to occur, such as the cæcum, the flexures, and the rectum, mechanical irritation of the mucous membrane is excited, and, ultimately, an inflammation arises, which may assume a severe form if the fæcal masses become decomposed and products of decomposition are formed, such as the corrosive ammoniacal gas.

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[1] Ueber die Diphtherie des Darms. Diss. Berolin. 1868.

[2] Ueber Diphtheritis des Darms im Puerperalfieber. Diss. Berolin. 1871.

The same explanation will apply also to those cases of intestinal diphtheria in which the characteristic infiltrations of diphtheria are found above pathological stenoses of the intestine. Thus, incarcerated hernia, invaginations, and tumors or cicatrices which narrow the lumen of the intestine, are sometimes attended by intestinal diphtheria above the constricted point.

Greater difficulty is experienced in explaining the genesis of the diphtheritic enteritis, which occurs in the course of various chronic severe exanthematic and septic diseases, such as carcinoma, tuberculosis, Bright's disease, typhoid and scarlet fevers, small-pox, septico-pyæmia, and especially puerperal fever.

In many of such cases we may suppose that the serious impairment of nutrition, resulting from the disease, produces a predisposition to enteritis,[2] the occurrence of which is specially favored, moreover, by the accumulation of blood in the pelvic organs and lower portions of the intestines, occasioned by the feeble action of the heart. Furthermore, a simple inflammation may very readily be converted into a diphtheritic one in the case of the intestinal mucous membrane, because the bowels very commonly contain sharply irritating products of decomposition. These products are re-enforced, in *septic diseases*, by the presence of the specific poison, which, being taken up by the bloodvessels in some other region of the body, is brought to the intestines, where it finds a habitat which is normally the seat of processes of decomposition, and is, for this reason, specially adapted to favor the further development and action of the septic matter.

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[1] Virchow's Archiv. V. S. 348 ff.

[2] See above, Etiology of Enteritis.

As regards the explanation of the diphtheritic intestinal affections, which occur in patients with *Bright's disease*, and particularly in cases of *uræmia*, it is to be borne in mind that the intestine is the only part where the *conversion of urea into carbonate of ammonia* takes place in this affection, and that this product of decomposition by itself, independently of putrefactive processes, has an undoubted tendency to produce diphtheritic inflammation of the intestinal mucous membrane.

In the intestinal diphtheria, finally, which occurs during the course of *puerperal fever*, there appears to be another etiological factor besides those mentioned, viz., the *direct extension* of the intense inflammatory process localized in the genital organs to the intestines by means of the peritoneum. At least this view is borne out by the experience of Freund, who, in eleven cases of puerperal diphtheria, found the diphtheritic lesions of mucous membrane nine times in the lower portions of the large intestine—that is, in those portions situated nearest to the genital organs.

The *symptoms* attending the above-mentioned forms of intestinal diphtheria are the same as those of the dysentery ; in other cases the disease runs its course without symptoms, if the inflammation be slight in extent, or if the fundamental affection be of an intense character, and rapidly terminate fatally. In such cases the pathologico-anatomical changes above-mentioned are found unexpectedly at the autopsy as casual accompaniments of the other lesions. We may expect to find them, however, if, during life, bloody, mucous, fatty, or purulent stools have been discharged, and still more certainly if other symptoms of dysentery, tenesmus, etc., have also been present; but these rather subjective symptoms of intestinal diphtheria are very frequently absent, or they may be disguised by those of the severe fundamental disease.

The *prognosis* in general depends, of course, chiefly upon the severity of the fundamental disease, and the prognosis of the *intestinal* affection in particular upon the intensity of the diphtheritic process, the existence of which, however, can only be suspected. At all events, the supervention of an intestinal diphtheria is invariably to be regarded as an extremely unsatisfactory complication.

The *treatment* is essentially the same as that of infective dysentery; and for this portion of the subject, as well as for the symptomatology, the reader is referred to the article on Dysentery in the first volume. As was mentioned in the consideration of the etiology, it is entirely proper, when the fundamental disease permits, to begin the treatment with a purgative, after which *tannin with opium*, and *injections with a solution of nitrate of silver*[1] may be used. At the same time *ice poultices* may be applied to the abdomen, and, in collapse, subcutaneous injections of camphor or ether may be resorted to. The *diet* should consist of nourishing food which makes as little fæces as possible: meat solution, milk, meat broths, etc.

Intestinal Ulcers.

Celsus, De medicina. Liber IV. Cap. XV., De torminibus: ("Intus intestina exulcerantur; ex his cruor manat, isque modo cum stercore aliquo semper liquido, modo cum quibusdam quasi mucosis excernitur, interdum simul quædam carnosa descendunt; frequens dejiciendi cupiditas dolorque in ano est—longoque tempore id malum, cum inveteraverit, aut tollit hominem, aut etiamsi finitur, excruciat.")—*Jacobus Fontanus*, J. F. Sanmaxitani primarii medico op. Colon. Allobrog. 1612. Medic. Pract. Lib. III. p. 180.—*Boneti*, Sepulchret. s. Anat. pract. Lib. III. Sect. XI.—*Joh. Conrad a Brunn*, Glandulæ duodeni seu Pancr. secund. etc. 1715. p. 112.—*Louis*, Mémoires de l'acad. Royale de med. 1767. cit. by Albers S. 8.—*Morgagni*, De sedibus et causis. etc. 1767. XXXI.—For additional old literature see *Plouquet*, Lit. med. digest. under Intestini Ulcera and Perforatio—*Lesser*, Entzündung und Verschwärung der Schleimhaut des Verdauungskanals. Berlin. 1830.—*Albers*, Die Darmgeschwüre. Leipzig. 1831.—PEPTIC ULCERS: *P. Rayer*, Observations sur les hæmorrhagies, etc. Archiv. génér. de med. 1825. VII. p. 166. (perforation from duodenal ulceration).—*Mayer*, Die Krankheiten des duodenums. 1844.—*Barker*, Perforating ulcer of the duodenum. Lancet. 1850.—*A. Claus*, Ueber spontane Darmperforationen. Diss. Inaug. Zürich. präs. Lebert. 1856.—*Wunderlich*, Fall von Duodenalgeschwür. S. 175, in his Handbuch der Pathol. u. Therap. 1856. Bd. III. 3.—

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[1] I generally use in dysentery a five-grain solution.

Friedreich, Ueber Amyloiddegeneration. Verhandl. des naturhist. medic. Ver. zu Heidelberg. 1858. V. S. 144. Ref. Schmidt's Jahrb. 103. S. 7.—*E. Wagner*, Fall von perforirendem Geschwür im Anfang des Jejunum. etc. Archiv für physiol. Heilkunde. 1858. N. F. II. S. 280.—*Müller*, Das corrosive Geschwür im Magen und Darmkanal. 1860.—*J. Krauss*, Das perforirende Geschwür im Duodenum. The best monograph on duodenal ulceration, containing a full compilation of the cases hitherto observed. 1865.—*G. Merkel*, Casuistischer Beitrag zur Entstehung des runden Magen- und Darmgeschwürs. Wiener medic. Presse. 1866. Nos. 30, 31. 1869. No. 39.—*Thierfelder*, Gastroduodenalfistel in Folge von corrosivem Magengeschwür. Archiv für klin. Medic. IV. 33. 1868.—*Aufrecht*, Amyloidgeschwüre des Darms. Berliner klin. Wochenschr. 1869. No. 30.—See also the literature on gastric ulceration.—*M. Zimmermann*, Ueber 2 Fälle von nekrosirender Enteritis bei Morbus maculosus Werlhofii. Archiv der Heilkunde. 1874. S. 167.—TUBERCULOUS ULCERS OF THE INTESTINES: *Rokitansky*, Pathol. Anat. III. 235. 1861.—*Virchow*, Onkologie. Bd. II. 1864–65. Vorlesung 21.—*Klebs*, Pathol. Anat. II. S. 257. 1869.—*A. C. Gerlach*, Ueber die Impfbarkeit der Tuberkulose und Perlsucht, etc. Virchow's Archiv. 51. S. 290. 1870.—*Zürn*, Zoopathol. u. zoophysiol. Unters. Stuttg. 1872.—*Friedländer*, Ueber locale Tuberkulose. Volkmann's Sammlung klinischer Vorträge. No. 64. 1873.—*Klebs*, Künstl. Erzeugung der Tuberkulose. Archiv für experim. Path. u. Pharmak. I. 1873. S. 163.—*Bollinger*, Ueber Impf- und Fütterungstuberkulose. ibid. S. 376. — *Rindfleisch*, Chronische Lungentuberk. Deutsches Archiv für klin. Med. 1874. XIII. S. 43.—(*Volz*, Die durch Kothsteine bedingte Entzündung des Wurmfortsatzes. Karlsruhe. 1846.)—*v. Bamberger*, Die Entzündungen in der rechten Fossa iliaca. Wiener medic. Wochenschrift. 1853. S. 369, containing a historical account of the disease, and a digest of the literature of the subject.—*Schnürer*, Ueber die Perforation des wurmförmigen Fortsatzes. 1854. Diss. Inaug. Erlangen (*Dittrich*).—*Leudet*, Recherches anatomo-pathologiques et cliniques sur l'ulceration et la perforation de l'appendice ileocoecal. Archiv général. de médecine. Aout. 1859. Ser. V. Tom. 14. p. 129.—*Rokitansky*, Darmentzündung. Pathol. Anat. 3. S. 202.—*W. Parker*, An operation for abscess of the appendix vermif. New York Med. Rec. II. 27.—The text-books of *Henoch*, *Wunderlich*, *Canstatt*, *Bamberger*, etc. —The very numerous reports of individual cases are not referred to, because they are not available for general purposes.

Although the possibility of intestinal ulceration was evidently admitted by ancient writers, as is shown by the passage from Celsus, quoted above, yet it was not until the practice of making autopsical examinations came to be more common that a knowledge of this exquisite pathologico-anatomical change in the intestinal canal was possible. Accordingly we find no correct description of intestinal ulcers until 1612, in the work of Jacobus

Fontanus, whose son had died of dysentery, and had been examined after death by Peter Bontamper. The latter found in the intestines more than two hundred round ulcers, intermingled with abscesses. Some of these ulcers had eaten through the entire thickness of the wall, while the intervening portions of the intestine presented a normal appearance. A more complete description of intestinal ulcers was given by Morgagni, who also treats of the products of secretion in intestinal ulcers, the masses of mucus, under the title of "corpora pinguia." The distinctions between the various kinds of intestinal ulcers, as regards their etiology and pathological anatomy, were not fully made until the present century, although Conrad von Brunn, in his work, issued in 1715, on "Glandulæ duodeni seu pancreas secundarium, etc.," described what was evidently a tuberculous ulceration of the intestines, especially of Peyer's patches, in a consumptive patient; and in 1767 Louis gave the first correct description of the intestinal lesions in typhoid fever.

The subject of intestinal ulcers, from a pathologico-anatomical point of view, has been treated so admirably by Rokitansky, that his descriptions have been largely drawn upon by later writers, and I shall also use them as the basis of my own. The *typhoid*, the *dysenteric*, and the rare *syphilitic* ulcers have already been described in other portions of this work, among the infective diseases, so that they may be excluded from present consideration. I wish to mention, however, the fact, as bearing upon the mode of formation of the other ulcers, to be presently considered, that the true syphilitic ulcers seem always to arise from gummy neoplasms, which, as Klebs supposes, originate in the submucosa and follicles. According to this writer, who has himself observed a very striking case of intestinal syphilis, these gummy neoplasms spread chiefly in depth, and laterally, and, after undergoing fatty degeneration and absorption of the gummy elements, result in the formation of radiated fibrous cicatrices; or the nodules may soften and break down into ulcers, which run round the wall of the intestine in an annular form in the same way as the tuberculous ulcers. These two forms of ulceration—the syphilitic and tuberculous—resemble each other also in their extension along the course of the lymphatic vessels of the serosa.

If these three forms of infective ulcers be excluded, the remaining forms may be divided into the following varieties:

1. Catarrhal ulcers.
2. Peptic ulcers.
3. Tuberculous ulcers.

The above classification is necessarily imperfect, because it takes into account only the etiology of the ulcers, and ignores the differences in their morbid anatomy. But in order to avoid repetitions, I shall consider the causes and anatomical appearances in a *single* chapter.

Etiology and Pathological Anatomy.

1. Catarrhal Ulcers.

In both acute and chronic intestinal catarrh small erosions occur, which begin with a loss of epithelium at particular points on the mucous membrane, and afterwards penetrate more deeply, so as to form what are known as *simple catarrhal ulcers.* In other cases, especially in chronic intestinal catarrh, the ulcers originate in a different way; the mucous membrane is not eroded from without, but at first looks as if covered with small collections of pus, which subsequently rupture into the cavity of the intestine. The ulcers thus formed enlarge peripherally and in depth, their edges become undermined, and they finally coalesce with each other. As soon as proliferation of the connective tissue has taken place, the islands of mucous membrane which remain intact upon the enlarged ulcerated surface are thrown forwards so as to give the appearance of polypoid growths, or, as Rokitansky has expressed it, "when considerable contraction of the base of the ulcer has taken place, the edges of the mucous membrane, the sinuses, and the islands of mucous membrane are pressed together in such a way as to present the appearance of a dense group of excrescences, between and at the bottom of which may be seen the cicatrix." The catarrhal ulcers present two courses: the ulceration may advance so as to erode the vessels, and finally produce perforation and fatal peritonitis, or the ulcer may heal, become cicatrized, and thus lead to a narrowing of the intestine followed by permanent obstruction. Certain portions of the intestines, the *cæcum*, the *vermiform appendix*, and the *rectum*, seem to be particularly liable to ulceration; and as the ulcers which occur in these parts are peculiar in their anatomical form, and in the course they pursue, we shall describe them separately.

The *cæcum*, in consequence of the angular course which the axis of the intestines takes at this point, acts as a natural resting-place for the food as it passes through the intestinal canal. Normally composed fæces may be here retained sufficiently long to become hardened or even converted into true fæcal calculi. These hardened fæcal masses, or indigestible substances swallowed in the food, such as kernels of grain, pins, pieces of bone, etc., may collect in the cæcum, particularly in the *vermiform appendix*, where they act as irritants to the mucous membrane and excite inflammation (*typhlitis*, especially *typhlitis stercoralis*), and may ultimately produce ulceration, partly from the mechanical pressure upon the intestinal wall (see above, Typhlitis). The ulceration may be of considerable superficial extent, and may penetrate so deeply as to perforate the bowel at one or more places. Perforation of the cæcal wall may be produced also in an opposite direction—that is, from without inwards—by an abscess originating in the cellular tissue surrounding the cæcum (perityphlitis). In the *inflammation and ulceration of the vermiform appendix* the causes are of a similar character. Here, also, it is usually hardened fæcal masses, intestinal calculi of an elongated bean-shaped form, corresponding to that of the vermiform appendix, foreign bodies, especially kernels of grain, etc., which first excite inflammation, and then a rapid necrosis of the intestinal wall. After the tissues have been destroyed in the base of the ulcer down to the serosa, the latter also becomes implicated in the ulcerative process, and it will now depend upon the rapidity with which the ulceration advances, whether the final perforation is to be preceded by the formation of adhesions to the neighboring parts or not. In the latter case the erosion of the serosa is immediately followed by the escape of the contents of the cæcum, and a usually fatal peritonitis ensues; in the former case the perforation is either entirely prevented, or the escaping contents of the bowel may collect between the adherent intestinal convolutions in the vicinity, or in the cellular tissue behind the cæcum, and give rise to iliac and lumbar abscesses, which may terminate in various ways. Under the most favorable circumstances the contents of these perityphlitic abscesses may be absorbed, or a cure may still be possible by the discharge of the pus externally, or through the intestine; less favorable, probably, is an evacuation through the bladder, uterus, etc., and most unfavorable, of course, a rupture of the encysted exudation into the free cavity of the peritoneum. When the suppurative process advances towards the external integuments, extensive gangrene may occur between the muscles both upwards and downwards, the pus may burrow along the retro-peritoneal cellular tissue into the pelvis, or up to the diaphragm, and perforate into the cavity of the chest, into the vena cava inferior, etc., or the case may terminate by a fatal pyæmia. The favorite situation for the perforation in the appendix is at the lower extremity, an entire section of which is sometimes sloughed off. In those cases in which the ulcerative process terminates unexpectedly before perforation of the serosa has occurred, in consequence, *e. g.*, of the escape of the impacted foreign body, cicatrization with contraction of the cæcum ensues, and the entire appendix may be converted in this way into a mere band.

In the rectum, also, ulcerative inflammation (*proctitis ulcerosa*) may occur as a result of chronic mechanical irritation from foreign bodies, impacted fæces, etc. Like typhlitis, this affection also readily leads to chronic inflammatory processes in the immediate neighborhood (*periproctitis*), with the formation of fistulæ, to girdle-shaped ulcerations, or to an extensive destruction of mucous membrane, with denudation of the thickened submucosa, followed by wasting and contraction of the rectum. The healing of such ulcers is very much delayed by the fact that the ulcerated and undermined mucous membrane is irritated by the fæcal masses, which are especially apt to accumulate in the lower part of the bowels. About the anus we find also hemorrhoidal swellings and ulcerations, which may be regarded partly as the cause, partly as the result of the ulcerative proctitis.

Essentially different, in an anatomical point of view, from these catarrhal ulcers of the intestines, although also like them resulting from inflammatory processes in the intestinal mucous membrane, are the *follicular ulcers.* Their form, at least in the early stage, is always round and funnel shaped, with marked thickening of the edges of the mucous membrane surrounding the ulcer. These characters may be explained by the mode in which the follicular ulcerations arise: the solitary follicles, especially in the large intestine, become swollen, as a result of catarrhal irritation, and the cellular elements accumulate in the reticulum, giving rise at first to nodules which project above the level of the mucous membrane; then the newly formed tissue elements become necrosed, in consequence of the mutual pressure of the cells upon each other; finally the apices of the follicular nodules give way, and ulcers are formed. The surrounding mucous membrane bends over downwards towards the base of the ulcer, "so that the orifices of the crypts look down into the same" (see the very instructive illustration in Rokitansky's Pathol. Anat. III. 1861. S. 226). As the suppurative process extends, particularly in the submucosa, and the tissue surrounding the follicles becomes destroyed, these small ulcers coalesce to form larger ones, and the undermined edges of mucous membrane project over the base of the ulcer, bleed, and become necrosed. Healing is possible by way of cicatrization, the borders of mucous membrane becoming applied to the base of the ulcer and gradually drawn together by the cicatricial tissue. Still this result is extremely rare, if the ulcerative process has once gained much headway. When, however, a follicular ulcer of some size does heal, cicatricial stenosis may result, followed by chronic constipation, just as in the case of simple catarrhal ulceration. The *situation* of the follicular ulcers is almost always in the *large intestine*, and they vary considerably in *number;* sometimes only a few follicles are thus affected, while in other cases the colon is actually crowded with ulcers. The most extensive ravages are found in the rectum and sigmoid flexure. The causes which give rise to the formation of follicular ulcers are the same as those of catarrh of the large intestine, if we except the follicular disease produced by dysenteric infection.

2. Peptic Ulcers.

Under this head I include those ulcerations of the intestinal canal which are produced by the action of the digestive juices. In the article on the round ulcer of the stomach and gastromalacia I have considered the conditions, which induce local self-digestion of the wall of the stomach. We there saw that the gastric ulcers were produced by a great variety of causes, all of which act by interrupting the local circulation, in consequence of which the acid gastric juice can impregnate the wall of the stomach to a greater depth and digest it, while, under normal circumstances, the blood circulating in the tissues renders them constantly alkaline, so as to protect them from self-digestion. Pavy's experiments (p. 266) apparently prove decisively that this protection is afforded by the alkalinity of the blood, which is incessantly renewed in consequence of the remarkable vascularity of the wall of the stomach. But if this were the only protection which the wall of the stomach possesses against self-digestion, I cannot see why the coats of the intestine, at least in the lower portions of the bowel, are not at all times exposed to self-digestion, because here we have juices which digest albumen and have an *alkaline* reaction, so that no chemical neutralization, such as takes place in the stomach, can be expected. Pavy's experiments have shown, moreover, that in the stomach it is not the epithelium which affords protection ; and if this fact apply also to the intestinal canal, the only explanation left is this, viz., *that the peptic destruction of the mucous membrane is prevented by the activity of the circulation in the wall of the intestine, by which the surplus digestive juices not used in transforming the chyme are absorbed before a digestive action can take place.*

Still it cannot be disputed that in the stomach a counteraction of the acidity of the gastric juice by the alkalinity of the blood is of the highest importance as a protection against self-digestion, because Pavy's experiments have shown that, when the acidity of the gastric juice is increased to a certain degree, the circulating blood, notwithstanding its alkalinity and its rapid renewal, is no longer able to check the self-digestion of the coats of the stomach.

The prototype of the peptic ulcers is the *perforating ulcer of the duodenum.* Most of what has been said in regard to the etiology of gastric ulcer will apply also to the causation of the duodenal ulcer, inasmuch as the latter is undoubtedly always due to the action of the acid gastric juice; on this point, therefore, the chapter on Gastric Ulcer is to be consulted. That here, also, the primary lesion is a local arrest of circulation, and that the formation of the ulcer at this point is a secondary and necessary result of the action of the digestive juice, has been made probable by the experiments of Panum, and has been directly demonstrated by Merkel's case, mentioned above (p. 200), which showed at the autopsy an embolized artery in the base of a duodenal ulcer. Another remarkable feature of the duodenal ulcer is its undeniable connection with burns of the skin. In *appearance* this ulcer resembles the gastric ulcer; it has usually a sloping shape, like a terrace, resulting from the fact that the loss of substance in the serosa is less than in the muscularis, and in the latter less than in the mucosa. The *edges* are sharply cut, not thickened, and in the older ulcers are indurated, in consequence of reactive inflammatory processes in the vicinity. The *base* shows no trace of suppuration, and is formed by the muscularis or the serosa according as the ulceration has penetrated more or less deeply. If the latter membrane be destroyed, perforation into the peritoneal cavity is produced, provided adhesion to some adjacent abdominal viscus, the *liver*, *gall-bladder*, or *pancreas* have not already taken place, in which case peritonitis will be avoided, as also when the ulcer penetrates directly into the dorsal region. In the latter case *external* perforation results,[1] or an extensive gangrene may supervene, which in one instance, described by Foerster and Krauss, extended upwards as far as the neck. Should the site of the ulcer fortunately become adherent to a hollow viscus, the rupture of the ulcer establishes an abnormal communication between two cavities; thus fistulæ between the duodenum and gall-bladder have been observed from this cause. No instance has yet been recorded of a gastro-duodenal fistula which could be proved to have resulted from the perforation of a duodenal ulcer; all the cases of abnormal communication between the stomach and duodenum, which have hitherto been noticed, have unquestionably proceeded from the perforation of a *gastric* ulcer. Very extraordinary malformations, which are exceedingly difficult to explain, such as division of the stomach into two separate cavities, have been noticed as a result of this lesion.

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[1] This location for the perforation has been observed on the right side in the seventh intercostal space.

Another frequent complication of the ulcer is the erosion of a large artery; in fact, all the arteries which could possibly be affected in this way (the pancreatico-duodenalis, the gastro-duodenalis, etc.), have been found eroded; in one case (Rayer) even the vena portæ.

If, on the other hand, the ulcer ultimately heals, it does so by the formation of a puckered cicatrix, which may produce the following results: stenosis of the lumen of the intestines, followed by dilatation of the intestine behind the obstruction or by dilatation of the stomach, occlusion of the pancreatic duct with atrophy of the pancreas, or of the ductus choledochus with stasis of the bile. In the latter case, jaundice is superadded to the other symptoms of the disease; but, except under these circumstances, jaundice is uncommon, probably because the mucous membrane of the duodenum in the neighborhood of the ulcer is usually in a normal condition.

Krauss's compilation of forty-seven cases showed that the ulcer was situated, in the great majority of cases, in the upper horizontal portion of the duodenum; occasionally one-half of the ulcer was situated in the stomach, the other half in the beginning of the duodenum. As regards the *number* of the ulcers, there is usually only one, but several are not unfrequently found, and it is not at all uncommon to find gastric and duodenal ulcers existing together.

Ulcer of the duodenum is a comparatively rare disease, the proportion to gastric ulcer being hardly one to thirty. The compilation of Krauss shows also that the lesion is most frequently met with at autopsies of persons between thirty and forty years of age, and that from sixty onwards it is rare, which only proves that this ulcer is, upon the whole, more frequent in early than in later life (see the statistics of gastric ulcer). As regards *sex* the relative number of duodenal ulcers, found by Krauss, in the two sexes is very remarkable ; out of sixty-four cases fifty-eight were men and six were women, a proportion of ten to one. This prevalence in the male sex is the more incomprehensible, because the frequency with which gastric ulceration depends upon chlorosis would lead us to expect that this cause would predispose also to the occurrence of duodenal ulceration.

Ulcers of the same characteristic form, and evidently likewise of a peptic origin, have in rare instances been found also in the lower portions of the intestine, viz., in the upper portion of the jejunum (Wagner), in its lower portion (Niemeyer), and in the colon (Lebert—Clauss). Finally, ulcers have been observed by Friedreich and Aufrecht in the ileum and colon in connection with amyloid degeneration of the vessels. These cases may be regarded as similar in character to the amyloid ulcers of the stomach noticed by Merkel, and are to be ascribed to the derangements of circulation resulting from the degeneration of the vessels.

When we consider that in the base and edges of *every* ulcer derangements of the circulation and probably, also, local stases are always present, it is obvious that even when the ulcer is not of hemorrhagic origin the digestive juices of the intestine must play an important part in the formation and enlargement of the ulcer. How important this share is I do not venture to decide ; further light must be obtained from experiments. In general, however, it is safe to say, even now, that the sharper the edges of the ulcer, the less the surrounding inflammation, the more we are justified in regarding the lesion as of peptic origin.

3. Tuberculous Ulcers.

The only way in which we can be absolutely certain in regard to the tuberculous nature of intestinal ulcers is to include under this head only those ulcers in whose immediate vicinity miliary tubercles can be detected. When the tubercles have undergone *complete* caseation, certainty in diagnosis is impossible, and the tuberculous character of the ulceration can be inferred with some degree of probability only when the other lesions in the body point in this direction.

Tuberculous ulceration in the intestines begins in the follicular apparatus, with swelling of the individual follicles and their vicinity from tuberculous deposit. The newly-formed cells become caseous, the superficial layer of the tubercle breaks down, and thus ulcerations arise of a round, funnel shape. The enlargement of these ulcers is probably produced by the constant formation in the base and edges of the ulcers of new nodules, which themselves caseate and ulcerate. This process of *extension*, to which the tuberculous ulcers of the intestine exhibit a marked tendency, takes place chiefly in a *transverse direction* (girdle shape), the same direction in which, as noted by Rindfleisch, the vessels of the intestines are distributed. The infiltration and necrosis may, however, advance also longitudinally, and, finally, the individual ulcers coalesce with each other; in this way may be explained, in part, the irregular dentate appearance of the edges of the ulcers. The ulceration extends also in depth, although usually the muscular coat appears to be covered by a thick layer of connective tissue. At all events, the destructive process advances in the muscularis slowly, but in the lymphatic vessels which pierce the muscularis less opposition is presented to the progress of the tuberculosis; and thus it is not uncommon to find tubercles ranged one after the other, like the links of a chain, from the base of the ulcer directly down to the serosa.

On inspection of the outer surface of the intestine, the *serosa* will be found to be opaque, or reddened, or to contain unmistakable miliary tubercles in its tissue opposite the sites of the ulcers. In by far the majority of cases these are the only lesions of the serosa; but occasionally the intestines are found to be glued to each other by a local adhesive peritonitis, so that when ulcerative destruction of the serosa occurs at a later period, the ingesta are discharged, not into the peritoneal cavity, but into a sac formed by the adherent intestines. Thus, in one of Rindfleisch's cases, a harmless rupture of this kind occurred at five different places in one portion of the intestines; in fact, perforation into the free cavity of the peritoneum, followed by perforation-peritonitis, is so very rare, that in the hundreds of patients with phthisis under my care in the Erlangen Hospital, during the past five years, I have seen this result only twice. Leudet, however, reports six cases of his own in which perforation of the vermiform appendix occurred in phthisical patients.

When the destructive process involves the walls of the vessels and the thrombi they may happen to contain, *hemorrhages* occur, and then the ulcer will be found covered with clots of blood or studded with hemorrhagic infiltrations.

Healing in the case of tuberculous ulcers is extremely rare, although the possibility of such an event is not to be absolutely excluded, for now and then we find more or less cicatrization, especially in the girdle-shaped ulcers, together with a flexion and narrowing of the bowel.

A further extension of the tuberculous process to parts outside of the intestine may take place through the lymphatic vessels. Thus we find the mesenteric glands enlarged, caseous, and often studded unmistakably with tubercles. That these processes—the tuberculous ulceration in the intestine, and the enlargement of the mesenteric glands—are causally related to each other, has been made still more probable by the recent investigations of Schüppel, Rindfleisch, and others, who have always been able to detect minute miliary tubercles in the swollen lymphatic glands.

The most frequent *situation* of tuberculous ulcers is in the lower end of the ileum. This may be their only location, or the process may extend upwards as far as the stomach and downwards as far as the rectum; occasionally the colon alone is affected, particularly the cæcum and vermiform appendix. In children the ulcerative process seems to attack the individual follicles of Peyer's patches separately, since in these cases minute funnel-shaped ulcerations are found upon the swollen patches corresponding to the site of the individual follicles.

Tuberculous ulceration is almost invariably accompanied by the signs of tuberculosis in other organs.

A primary occurrence of this lesion is entirely rejected by some pathologists; I have myself never seen intestinal tuberculosis unless accompanied by tuberculosis elsewhere. It is most usually associated, as is well known, with marked pulmonary tuberculosis, which in adults is the predominant affection, but in children is less developed.

The *causes* of the tuberculous ulceration are of the same nature as those of tuberculosis in general; but this is a subject which cannot be fully considered here. It may be remarked, however, in reference to the genesis of intestinal tuberculosis, that, in the words of Virchow, "the predisposition to tuberculosis, the hereditary vulnerability, resides in the tissues, and that the younger and more incompletely developed these are, the more readily will the vulnerability manifest itself in the presence of exciting causes." This may account for the special frequency of intestinal tuberculosis in childhood, a period during which the intestines are subjected to very numerous hurtful influences, and are particularly liable to be overtaxed by indigestible food.

Another question of great importance is, whether tuberculosis of the intestine may not be induced by the introduction of a tuberculous virus from sources external to the body.

Although I have thus far carefully refrained from committing myself to any theory of the genesis of intestinal tuberculosis, and although all the evidence that has been advanced during the past eight years in favor of the inoculation and infection theory of tuberculosis seems to me far from convincing, yet the experiments which have been made by feeding animals with tuberculous matters are so important in their bearing upon the causation of tuberculosis, and especially intestinal tuberculosis in the human subject, that I do not see how I can well avoid a brief reference to them.

In the first place, Klebs has supposed that the ulcerations found in the intestines of consumptive patients might be explained by the swallowing of tuberculous sputa. This opinion he based upon a very striking case, in which the lesions found at the autopsy could be accounted for in this way. In addition to the chronic pulmonary tuberculosis, two tuberculous ulcers were discovered in the stomach together with a secondary tuberculosis of the peritoneum. He supposed, therefore, that the swallowed sputa might excite tuberculosis of the follicles of the small intestine, and in this way additional infective material might be produced, which, being carried into the lower portions of the intestines, would account for the more marked affection of the large intestine. This hypothesis received further support from the experiments of Malin, Parrot, and Bouley, who found that animals fed with the sputa of consumptives died tuberculous; while Chauveau, Bollinger, and others, succeeded in directly producing intestinal ulcerations in addition to general tuberculosis by feeding animals with pieces of tuberculous lungs, with the meat of cattle suffering from "pearl-disease," etc. If, as these experiments seemed to prove, it is possible for infection to occur through the intestinal canal, it evidently follows that the use of *meat* from tuberculous cattle is at least attended with risk. The still greater danger to the health of the community arising from the use of *milk* from tuberculous cows has been shown by the experiments of A. C. Gerlach, Zürn, and Klebs. The last observer has come to the conclusion "that the milk of tuberculous cows has the power to excite tuberculosis in various animals, and that the disease usually begins with intestinal catarrh, then extends to the mesenteric glands, liver, and spleen, and finally to diffuse miliary tuberculosis of the thoracic organs—in fact, runs the same course as scrofula and secondary tuberculosis in the human subject." The results of these experiments afford a natural explanation of the occurrence of tuberculosis in children without any hereditary predisposition, but who have been fed with milk from tuberculous cows, or have had consumptive wet-nurses.

In the present unsettled state of the question in regard to the pathologico-anatomical identity of human and animal tuberculosis, and in the absence of a sufficient number of feeding experiments to decide so eminently important a question, all that can be said is, that the possibility of an infection by tuberculous virus introduced into the intestine from without can neither be positively affirmed nor denied, and that further experiments on a most extensive scale are extremely desirable. The etiology of human tuberculosis, and particularly the tuberculosis of children, it seems to me, can be profitably studied in regard to this question only by means of large statistics, by a careful supervision of dairies, etc.

Symptomatology.

The clinical symptoms produced by an ulcerative process in the intestines are by no means characteristic, and so long as the ulcer is not directly accessible to inspection, or is unaccompanied by unequivocal secondary results of a severe character, particularly perforation into the peritoneal cavity, the diagnosis in the great majority of cases will be necessarily uncertain, and will scarcely rise above a mere suspicion. It is not to be wondered at, therefore, that ulcers are often found in the intestines without the slightest evidence of their existence during life. On the other hand, in diseases like typhoid fever and pulmonary tuberculosis, which are attended by ulceration of the intestines, the severity of the intestinal symptoms—for instance, the amount of *diarrhœa*—is a very unsafe guide in regard to the amount of ulceration. The following cases, which were observed recently in immediate succession, illustrate this point:

CASE 1.—During the last six months, *severe pains in the abdomen, persistent diarrhœa*, and a corresponding degree of emaciation. For the last three months, symptoms of condensation of both apices. The autopsy revealed two small ulcers, as large as lentil seeds, in the large and small intestines, together with advanced tuberculosis of the lungs.

CASE 2.—*Typhoid fever*, with persistent diarrhœa; from four to six discharges daily. After death, which occurred at the height of the typhoid process, *a single ulcer was found upon the otherwise uninflamed small intestine.*

On the other hand, cases not unfrequently occur in which there has been constipation instead of diarrhœa, and yet the autopsy shows a diffuse ulcerative process in the large and small intestines. Such occurrences are obviously to be explained by the fact that the diarrhœa depends chiefly upon the increase of the peristaltic movements of the large intestine, particularly its lower portion. Normally, the chyle passes through the small intestine very rapidly—in fact, will make its appearance in a fistula of the ascending colon within a few hours; hence, as far as the causation of diarrhœa is concerned, a greater or a less delay in the ileum or cæcum, where the ulcers are chiefly located, is evidently of comparatively little importance. A part of the causation of the diarrhœa must, of course, be sought for in the ulcers themselves—at least, as soon as they have acquired any considerable size. Aside from the irritation of the nerves in the base of the ulcer, by which increased peristalsis may possibly be excited, the loss of absorptive power produced by the destruction of the absorbent vessels in the ulcers will of itself occasion an excessive flow of the intestinal fluids into the lower parts of the bowel, and may thus possibly produce diarrhœa. This factor is to be taken into account particularly in the explanation of the persistent diarrhœa in the intestinal tuberculosis of children (tabes mesenterica). Generally, however, when diarrhœa accompanies ulceration of the intestines, it is produced by a more or less extensive catarrh, and it is always to this, rather than to the special intensity of the ulcerative process, that a persistent diarrhœa under these circumstances is to be ascribed.

Still more important, in reference to the diagnosis than the frequency, is the *character of the stools.* The frequent presence of *blood* is decidedly suspicious, since bloody stools are very rare during the course of simple catarrhal diarrhœa, and never occur under these circumstances unless the inflammation is very acute or there is an abnormal lacerability of the intestinal vessels. Besides enteritis, the other causes of hemorrhage must of course be excluded, especially portal congestion, hemorrhoids, neoplasms of the intestines, etc. In the perforating ulcers of the duodenum profuse hemorrhage is a very common symptom, occurring, according to Krauss, in one-half the cases. The blood which has escaped from the eroded vessel in the ulcer may be discharged upwards, downwards, or in both directions at the same time ; in fact, hematemesis, in addition to the bloody stools, has very generally been reported as one of the symptoms of the duodenal ulcer. The blood varies in color from its natural red to a dark or even black hue, according to its quantity and the rapidity of its discharge. When the ulcers are situated in the lower parts of the intestinal canal, where the fæcal masses are already formed, the latter are discharged with an external coating of blood, but when the hemorrhage proceeds from a higher source the blood is intimately mixed with the fæces. In tuberculous ulceration, this admixture is, in my experience, rather unusual, probably because, as happens also in the case of follicular ulcers, a slight coating of blood over the fæcal masses is either rubbed off by the intestinal wall on their way to the sphincter, or else it is changed by the digestive processes in the large intestine so as to become unrecognizable.

Pus may be expected in the stools only when the ulcers are situated in the lowermost parts of the large intestine; in the upper portions there can hardly be said to be an actual purulent discharge upon the ulcerated surface, because the digestive juices not only constantly wash the base of the ulcer and carry off the pus along with them, but also by their peptic action check its formation. At the same time, it should not be forgotten that the cells of the intestinal juice resemble pus cells so closely that it is difficult to decide whether the cells mixed with the fæces are pus corpuscles or not.

Particularly characteristic of ulceration is the presence in the discharges of masses of *mucus*, which represent the form of the ulcer—the "*transparent particles of mucus resembling frog-spawn or boiled sago grains*" (Bamberger). These masses are probably produced by the accumulation of mucus in the funnel-shaped ulcers, which arise from the ulceration of the follicles, the casts thus formed being thrown off entire. At all events, the secretion comes from the Lieberkühnian follicles, and probably from those in which, as before described, the orifices look down towards the base of the ulcer. A large part of these "mucous masses" are, however, not mucus at all, but imperfectly digested starch, as Virchow[1] pointed out some time ago.

Vomiting occurs as a direct result of the ulcerative process only in the case of a duodenal ulcer, and is then due to the proximity of the ulcer to the pylorus. The pylorus may thus be kept in a state of constant contraction, and vomiting movements on the part of the stomach may, in this way, be excited at intervals. This symptom may occur, however, as a complication of the ulcerative process, wherever situated, if the inflammation has excited irritation of the peritoneum, vomiting being, as is well known, one of the most frequent symptoms of such irritation.

Icterus, like the vomiting, is peculiar to ulcers of the duodenum, and, as we have seen above, is an extremely rare symptom. If a long-continued jaundice occur during the course of a duodenal ulcer, it is probably due to a closure of the ductus choledochus by a permanent obstruction, generally a cicatrix.

In a case observed by Barker, the jaundice was the result of the extension of a catarrhal inflammation of the gall-bladder, which communicated with a perforating ulcer of the duodenum.

In rare cases, icterus may occur in connection with an ulcerative process lower in the intestines, in consequence of the extension of the accompanying catarrh to the duodenum, or the jaundice may be a symptom of pyæmia, resulting from the suppuration produced by a perforating intestinal ulceration.

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[1] Virchow's Archiv. V. S. 329.

As regards the pain, which is said to accompany the formation of intestinal ulcers, its wholly subjective and very ambiguous character renders it of less symptomatic importance than has commonly been ascribed to it. But if the pain can be definitely located, if it be considerably aggravated by slight pressure and be attended by vomiting, the symptom ought not to be neglected, because now an irritation of the peritoneum by the ulcer may be regarded as certain. In ulcerations of the cæcum, or vermiform appendix, this result is particularly common; in fact, it is the occurrence of sharp peritoneal pains which usually calls attention to the ulcerative process, the long previous existence of which had been concealed by the absence of symptoms. The *character of the pain* is undoubtedly influenced by the locality where the ulcers are situated. Thus the duodenal ulcer is accompanied by *cardialgic attacks*, which usually come on some time after eating (though not very much later than in gastric ulcer), and differ in no respect from the cardialgias produced by morbid conditions of the stomach. The pains in these cases are sometimes paroxysmal, sometimes continuous; usually they are aggravated by pressure upon the epigastrium, and occasionally are more severe during the night (Mayer, p. 106). Usually, eating excites the pain, and vomiting relieves it, but sometimes eating gives striking relief. Ulcers in other parts of the intestines may give rise to *colicky* pains and rectal ulcers to *tenesmus*—that peculiar bearing-down feeling which conveys to the patient the impression as if something were sticking in the rectum and had to be gotten rid of, and which is accompanied by a convulsive contraction of the sphincter.

Whether the fever attending the ulcerative process depends upon the latter or upon the fundamental disease, it is difficult to decide with certainty, and yet the absence of fever in cases of ulcer of the duodenum would seem to show that this symptom is due entirely to the fundamental disease—the tuberculosis or the intestinal catarrh. When fever, increased pain, and vomiting set in during the course of a hitherto afebrile disease, attended by symptoms of intestinal ulceration, such a combination of symptoms points to the onset of peritonitis.

When the ulcerative process is of a very diffuse character, and continues for a considerable time, the *general health* becomes impaired, though here, also, it is the fundamental disease which is chiefly responsible for the deterioration of health. When we bear in mind, however, that in cases of intestinal ulceration the absorption of the digested food in the intestine is hindered by the destruction of a certain portion of the lymphatics, and that this deficient absorption, as recent investigations in regard to the digestive process in the colon have shown, must exert an important influence upon the nutrition of the entire body ; when we consider, moreover, that, before the food can be absorbed, it is discharged from the body in an imperfectly digested form, either by vomiting, when the ulcer is located in the duodenum, or by diarrhœa, when the lesion is situated lower down, it is difficult to see how an ulcerative process can continue for any considerable time in the intestinal canal without of itself seriously interfering with nutrition of the body. Hence, the great appropriateness of the term—tabes mesenterica—which the older writers employed for certain forms of intestinal ulceration.

In some cases of intestinal ulcers a *tumor* can be distinctly felt, or even seen, in the region of the ulcer. In the case of the perforating duodenal ulcer, the tumor may be formed by the pancreas which has become secondarily enlarged and indurated (Wunderlich); in ulcerations of the cæcum, or vermiform appendix, by the products of the local peritonitis, which either precedes the occurrence of perforation, or serves as a safeguard against it. In the typhlitic ulcerations the tumor is of considerable size, because, in addition to the peritoneal exudation and inflammatory thickening of the coats of the bowel, we have also an accumulation of fæces caused by paresis of the musculature of the cæcum. The tumor in these cases is situated in the right ileo-inguinal region, just above Poupart's ligament, with its long axis directed inwards and downwards towards the median line. It is frequently of quite a solid consistence, very tender to the touch, sometimes of a sharply defined, at other times of a rather diffuse outline, and dull or dull-tympanitic on percussion. The disappearance of the tumor takes place very gradually, often occupying weeks or months, unless the occurrence of a general peritonitis, or the escape of air into the peritoneal cavity, conceals its examination. When the cellular tissue, which lies behind the cæcum upon the fascia iliaca, is implicated in the inflammatory process, or when the peritoneal exudation in the vicinity of the cæcum presses upon the ilio-psoas muscle, the free contractions of this muscle will be impeded, and the lumbar nerves, which pass through this muscle, will be compressed. This is the explanation of the pains and abnormal sensations (formication, etc.), which occasionally occur in the lower extremities and genitalia in typhlitis or perityphlitis, and also of the difficulty in flexing the right thigh upon the abdomen. When the exudation presses upon the iliac vein, thrombosis may be produced in this vessel, and thus give rise to œdema in the right leg.

It must be evident, from our consideration of the subject thus far, that *the symptoms which attend the ulcerative process in the intestines are in no respect pathognomonic ;* for the symptoms last mentioned, which are of a much more objective and distinctive character than those earlier described, belong, in reality, not so much to the ulcerative process itself, as to the further changes which result from it.

When the ulceration of the intestinal wall has extended to the peritoneum, perforation of the latter and the occurrence of *general peritonitis* will ensue, unless prevented by adhesive inflammation or the other causes above described. Usually this result is accompanied by an escape of air, as shown by the disappearance of the liver dulness, etc. Under special circumstances, however, the escape of air into the peritoneal cavity fails to occur, although the perforation has taken place at a part where the serosa surrounds the entire circumference of the intestine—for instance, when the bowel is perforated posteriorly and is at the same time more or less immovable. The following case,[1] recently observed by me, may serve as an illustration of this point :

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[1] Berichte der Jenaer Klinik. S. 44.

A woman, suffering from stenosis and insufficiency of the mitral valve, was attacked with a chill of an hour's duration; the expectoration was bloody, and examination of the chest revealed dulness over the right inferior lobe. Vomiting set in at once; temperature, 102° F.; intense pain in the epigastrium, and distention of the abdomen. The dulness on percussion extended as far as the extremity of the ensiform cartilage and to the curve of the ribs. A diagnosis was accordingly made of hemorrhagic infarction of the lung, complicated, in all probability, with peritonitis; but it was impossible to determine the cause of the latter. Death took place thirty hours after the onset of the acute symptoms.

The *autopsy* cleared up the obscurity of the case. In addition to the stenosis and insufficiency of the mitralis, and the hemorrhagic infarction, the following lesions were found in the abdominal cavity: a considerable quantity of reddish-yellow, turbid fluid; a loose, reticulated, grayish-yellow deposit of fibrin covering the intestines; and, *on the posterior aspect of the duodenum, near its commencement, an ulcer, two ctm. in length by half a ctm. in breadth, with a slightly raised border, which ulcer had produced a perforation of the size of a lentil near the attachment of the duodenum.*

In consequence of the entire absence during life of all indications of a severe affection of the digestive tract, the existence of the duodenal ulcer had not been suspected, nor had there been the usual signs of the perforation of an air-containing abdominal viscus, such as the displacement of the abdominal organs by the pressure of the air. The failure of the gas to escape, even after the lapse of thirty hours, might be readily explained by the locality of the perforation, the intestine being here less mobile than it is elsewhere, while in the recumbent position of the patient the semi-solid fæces would naturally sink downwards and backwards, and the gas would accumulate anteriorly (*i.e.*, superiorly).

In those parts of the intestines which are not covered by a serous coat, viz., the posterior wall of the second and third divisions of the duodenum and that of the ascending and descending colon, ulcerative perforation may occur without implicating the peritoneum; in these cases the ulcerative process extends, per contiguitatem, to the cellular tissue about the large vessels on the posterior wall of the abdomen, and particularly to the cellular tissue in the vicinity of the kidneys and quadratus lumborum.

As an aid to the diagnosis in particular cases, we give below a brief sketch of the various groups of symptoms which are produced, according as the disease is modified by its locality and mode of origin.

The *duodenal ulcer* in many cases first manifests itself by the symptoms which immediately usher in the fatal result, the hæmatemesis or the rapidly-fatal perforation taking place in the midst of apparent health. Usually, however, even when the duodenal disease runs a rapid course, careful inquiry into the general health of the patient will show that he has previously suffered from slight dyspeptic symptoms—oppression in the epigastrium and uncomfortable feelings after meals. In the more gradually developed cases the symptoms are very similar to those of simple gastric ulcer. The pains are sometimes confined to the right hypochondrium ; in some cases they are of a continuous character ; in others they occur in paroxysms after eating ; or they are increased by external pressure. Vomiting of the contents of the stomach, with or without admixture of blood, appears to be comparatively infrequent, unless the duodenal ulceration have superinduced a dilatation of the stomach. Icterus must be regarded as a rare complication. The fatal catastrophe is ushered in either by severe peritoneal pain, followed by the symptoms of perforation, or by vomiting and purging of blood. If the blood be retained, instead of being discharged, death may occur rapidly, with the symptoms of internal hemorrhage. Only in very exceptional cases does cicatrization of the ulcer take place with the above-mentioned after-symptoms—dilatation of the stomach or chronic icterus. Derangement of the general health is also very rare. The diagnosis is always doubtful, and a distinction from gastric ulcer is well-nigh impossible. The situation of the ulceration in the duodenum may, however, be *suspected*, if the pains are felt exclusively in the right hypochondrium, if the blood passes off in a downward direction, and if the history of the case shows the previous occurrence of an extensive burn of the skin.

Ulceration of the cæcum or vermiform appendix gives rise, as is well known, to very striking symptoms. In some cases the attack occurs suddenly without premonitory symptoms, in others it is preceded by constipation, dull pain in the abdomen, attacks of colic, and other like abdominal derangements of a general nature. The immediate attack is ushered in by severe pain limited to the right side of the abdomen, increased by motion, even by breathing, and aggravated to its greatest intensity by pressure upon the right inguinal region. Palpation in this location reveals a tumor with the characteristics described above, composed partly of impacted fæces and partly of inflammatory exudation ; the percussion note is dull, or, at least, dull tympanitic, and the movements of the right thigh are painful. The occurrence of peritonitis will be shown by fever, vomiting (sometimes of fæces), singultus, and an increasing intensity of the pain, while this diagnosis will be placed beyond doubt by the collapse of the patient, the coldness of the extremities, the meteorism, and the dyspnœa. In the milder cases, in which a circumscribed peritonitis occurs without rupture, or a rupture takes place without the escape of any considerable quantity of the contents of the intestine, healing is still possible either by the exudation becoming encysted and gradually absorbed, or by the intestine, particularly the cæcum, becoming again permeable, and regaining its former contractile power. In the severe cases the circumscribed inflammation extends to the surrounding parts —the psoas muscle or the cellular tissue around the kidneys and rectum (perinephritis, or proctitis)—or it may go on to produce gangrene, etc. (see above).

If the encysted exudation ruptures into one of the hollow abdominal viscera a complete cure may even now be effected, as recently happened in one of my cases, in which the abscess in the neighborhood of the vermiform appendix discharged into the rectum, and the escape of fæces into the cavity of the abscess was prevented probably by the valvular nature of the perforation and by the pressure of the surrounding intestines upon the wall of the abscess. On the other hand, death may result from the supervention of pyæmia, or from a subsequent rupture of the encysted exudation into the free cavity of the peritoneum. In the latter case the same symptoms occur as when the perforation of the cæcum or vermiform appendix is immediately followed by the symptoms of a general peritonitis.

Usually it is impossible to determine whether the cæcum or vermiform appendix is the starting-point of these urgent symptoms. In a doubtful case it is always more probable that the ulceration affects the vermiform appendix, since, according to Bamberger, this portion of the intestine is the seat of the ulceration in about four-fifths of the cases of fatal peritonitis, starting in the cæcal region. In general this diagnosis, as opposed to that of an ulcerative typhlitis, will be more certain if the history of the case shows that the patient has been attacked *suddenly* in the midst of health, and if the presence of a solid tumor in the cæcal region is discovered, not at the very outset of the attack, but only after the development of the circumscribed peritonitis. In typhlitis the tumor is to a large degree formed by an accumulation of fæces, and will therefore be detected at the start, while the ulceration and perforation of the cæcum are preceded by symptoms of functional derangement of this part of the intestine, fæcal impaction, attacks of colic and tenderness in the right inguinal region.

Ulcerations of the rectum, on account of their situation where they can be directly examined by the finger, bougie, and speculum, are more readily diagnosticated than ulcers in other parts of the intestines. They are attended by tenesmus, pain in defecation, and by the discharge of pus and blood, which present an unaltered appearance because they come from points near the outlet of the intestinal canal. The bowels are in some cases constipated in consequence of the accumulation of the fæces above the ulcerations, in other cases there is diarrhœa. These symptoms may, of course, all be absent, and then the doubt is to be cleared up only by the discovery of fixed points of pain on the introduction of the finger or bougie, which, when withdrawn, are found covered with blood or pus. If the ulceration result in perforation, the rupture may take place into the bladder, vagina, or externally; and in the latter case a rectal fistula will be formed.

The diagnosis of *follicular ulcers*, higher up in the different portions of the colon, is less certain. In protracted cases of intestinal catarrh, attended by blood and mucus in the stools, particularly the above-mentioned lumps of mucus resembling sago grains, these follicular ulcers are to be borne in mind. The marasmus, which sometimes occurs during this variety of intestinal ulceration, is to be ascribed to the imperfect absorption of the food by the ulcerated intestine. The occurrence of severe pain, increased by pressure at certain places in the course of the colon, points to the possible supervention of peritonitis. Although this event is rare in follicular ulceration, it is to be apprehended, if the tender region be occupied by a tumor resembling that of typhlitis. The stools vary in character; attacks of diarrhœa alternate with the discharge of scybalæ covered with blood and mucus, and obstinate constipation ensues when the ulcers heal, and leave cicatrices which stenose the lumen of the intestine. The intercurrent attacks of diarrhœa in these cases are due to the catarrh excited by the irritating solid fæcal masses, which occasionally adhere to the wall of the intestine in such a way as to leave a central canal through which the thinner fæces are discharged. Extensive follicular ulceration, particularly in children, may terminate fatally with symptoms of marasmus, especially if the appetite, which has hitherto been maintained, be finally lost.

It is usually very difficult to decide whether the suspected intestinal ulcers in children are of a *tuberculous* nature or not. Tuberculous ulceration will be indicated by the more intense and more continuous fever, the greater emaciation, the evidence of condensations in the lungs, and of an hereditary predisposition, and, finally, as may be possible in some instances, by the palpation through the abdominal wall of swollen tuberculous mesenteric glands. These glands present the feeling of comparatively immobile tumors about the size of a nut; but unfortunately they frequently escape detection in consequence of the tympanitic distention of the abdomen by gas, which renders any palpation of the deeper lying parts impossible. Notwithstanding the frequency of tuberculous ulcerations during the course of phthisis, it is difficult to make a *certain* diagnosis of their existence during life. To be sure, the presence of continuous diarrhœa, the admixture of traces of blood in the fæces, and the tenderness of the abdomen at certain places, especially the ileo-cæcal region, may induce a suspicion that intestinal tuberculosis has been superadded to the pulmonary affection; but how little reliance can be placed upon these symptoms must be sufficiently evident from the preceding considerations. Not until a severe intestinal hemorrhage or a perforation-peritonitis—both of which, however, are very rare complications of tuberculous ulceration of the intestines—change the scene, and almost invariably terminate it, can the diagnosis be made with much certainty. A cure is never to be expected; the partial cicatrizations lead to stenosis and flexions of the bowel with chronic constipation, while the ulcers, cicatrices, and tuberculosis of the mesenteric glands involve a permanent loss of absorbent vessels, so that the nutrition is always impaired and the emaciation in consumptives materially promoted by the intestinal tuberculosis.

Prognosis.

The *prognosis* is unfavorable in every form of intestinal ulceration, and particularly so in *tuberculous* ulceration of the intestinal glands. But even the simple catarrhal, and especially the *follicular ulcers*, when they are diffusely spread, may prove fatal through the exhaustion produced by the intestinal affection, not to speak of the risk from perforation. When they are of moderate extent and have lasted but a short time, they may heal without leaving any traces; but if the loss of tissue has been considerable, cicatricial structures are left after healing, which give rise to obstinate constipation.

Ulcers in the lower part of the *rectum* are of course most favorably situated for relief by medical treatment; but here, also, if the ulcerations be extensive, there is always danger that the cure will be marred by the subsequent formation of cicatricial strictures.

In *ulceration of the cæcum*, healing may still occur even when the lesion is of considerable extent, and in the vermiform appendix the occurrence of perforation may be prevented by the formation of adhesions. In fact, there may finally be a complete cure in cases of deep ulceration as a result of the local peritonitis developed around the cæcum. The dangers, however, in ulceration of these parts, as shown by the previous description of the disease, are too serious to permit, as a rule, any other than a doubtful prognosis.

The same remark will apply, with perhaps even greater force, to cases of *ulcer of the duodenum*, because here cicatrization is an extremely rare event, half of these cases, according to Krauss's compilation, terminating with perforation, and about one-fourth with profuse hemorrhage.

Treatment.

The *prophylaxis* requires that intestinal catarrh should be avoided as far as possible, and especially that it should never be neglected when it once occurs. The latter precaution is particularly necessary in the intestinal catarrhs of young children, in which a careful regulation of the diet will usually constitute the most important part of the treatment (see above). In view of the still unsettled state of the question, whether intestinal tuberculosis in children can be produced by the milk of tuberculous cows, it will be well, at all events, to observe the greatest caution in the use of cow's milk, and to allow it to be used only after it has been boiled. When there is reason to suppose that the ulcerative process is caused by an accumulation of fæces or by indigestible substances which have been swallowed, the first indication will be to remove these masses by means of injections or by a laxative. The best laxative for this purpose is castor-oil, either pure or in emulsion, because, being the mildest of purgatives, it is least likely to injure the inflamed mucous membrane. The removal of the fæcal masses is also urgently indicated during the further course of the treatment for the purpose of relieving the ulcers of an important obstacle to their healing (see above).

The direct treatment of the ulcers themselves is attended with great difficulties, because, except in the case of rectal ulcerations, the lesions are inaccessible to energetic treatment. The prescription of *nitrate of silver* (a grain and a half in six ounces of water), *tannin* (thirty grains in four ounces), *bismuth*, *acetate of lead*, etc., is useless, except for the purpose of relieving the complicating intestinal catarrh ; little can be expected from the internal use of these remedies, as far as any alterative action upon the ulcers is concerned. A better result may be expected from the employment of astringent enemeta, particularly those containing *nitrate of silver*, which can be injected as strong as one part to the hundred without exciting pain. Sulphate of zinc, or tannic acid, may be used in the same way. The astringent injections, in order to have any effect, must be given when the intestine is empty, and should therefore be preceded by an enema of water to clear the parts. The more accessible rectal ulcers obviously require to be treated in accordance with the same rules which govern the treatment of ulcers situated on exposed parts of the body. In other cases—since the lesions are almost always situated so high up that the remedies used to counteract the ulcerative process do not reach the parts where their effect is required, or at least do so in far too small a quantity—all that can be done is to remove from the surface of the ulcer everything that can check its natural tendency to healing. Above all is it important, as already remarked, to guard against irritation of the ulcerated surface by fæcal masses lying upon it; and therefore, during the course of treatment, the patient may be allowed to drink mild cathartic mineral waters (Marienbad, Kissengen, etc.), or, when the ulcers are situated in the large intestine, cold-water enemata, or, still better, irrigations may be used. In the latter case a little carbolic acid may be added to the water, for the purpose of checking, in some degree, the processes of decomposition in the large intestine, particularly as regards their action upon the ulcerated surface. The diet should be such as will not form large quantities of fæces, and all articles of food (large pieces of cheese, or fruit, etc.), which can mechanically irritate the ulcerated wall of the intestine, are to be carefully avoided. In general, therefore, the diet should consist of meat and milk, while vegetables, and especially brown bread, are to be used very sparingly; in the ulcerative intestinal catarrh of children essentially the same diet is to be selected which was mentioned for the treatment of cholera infantum.

When an *ulcer of the duodenum* can be diagnosticated with some probability, it is best to send the patient to Carlsbad, and to treat the case in accordance with the rules laid down for gastric ulcer; at the same time it will almost always be impossible to tell absolutely whether the case in question is one of gastric ulcer or one of ulceration of the duodenum.

Certain symptoms of intestinal ulceration, when they are very severe, require special therapeutic measures.

The pain connected with the ulcerative process is occasionally so distressing, when there is marked peritoneal irritation, or when regular cardialgic attacks occur, as in the duodenal ulcer, as to require medical interference. In such cases the administration of narcotics, particularly opium, is the more indicated from the fact that a second indication is thereby fulfilled, viz., the quieting of the violent peristaltic movements, which keep up the pains. The extract of belladonna is also to be recommended, on account of its special power to relieve the hyperæsthesia and the abnormal peristalsis. *Vomiting*, likewise, requires the use of narcotics, whether it be conditioned by the locality occupied by the ulcer, as in the ulcer of the duodenum, or whether it be symptomatic of a complicating peritonitis. In the latter case cold may also be employed externally, in the form of an ice-bag, over the painful part of the abdomen. To quench thirst, ice-water, or pieces of ice, are allowable under all circumstances, and iced champagne upon the occurrence of collapse.

We have seen above that when the bowels are sluggish and the temporarily retained fæces are evidently causing an irritation of the ulcerated surface, the indicatio morbi requires the regular use of cathartic mineral waters, irrigations, etc. But we have seen also that ulcerations of the intestine may be accompanied by diarrhœa. If the latter be so considerable as to affect the general health of the patient, and if the case be complicated by an intestinal catarrh, as shown by the character of the stools, and by the extension of the tenderness on pressure over a large portion of the abdomen, then, in place of the measures above mentioned, remedies to control the diarrhœa are required: red wine, barley-water, astringents, such as tannin, nitrate of silver, etc., columbo, and especially opium, which in the catarrhs of the large intestine is best given by way of enema (decoction of salep with fifteen minims of laudanum).

When the increased pain and vomiting and the occurrence of collapse indicate the supervention of peritonitis, the use of opium then becomes indispensable, and has long been regarded as the remedy *par excellence.* At the very outset of the peritonitis, however, if the commencing inflammation of the peritoneum seems to have been excited by a marked constipation of the bowels in connection with the ulcer, it may be proper to begin the treatment with a dose of castor-oil in place of the opium. But when the peritonitis has once become unmistakable, the administration of opium is the only rational treatment, because by this means alone can we prevent the rupture of the newly-formed adhesions, quiet the peristaltic movements, which play an important part in the spread of the inflammation, and, in a word, control the intensity and extension of the peritonitis. The attainment of this object is in such cases incomparably more important than an evacuation of the bowels. In fact, the constipation is due in part to the inflammatory infiltration of the wall of the intestine, and, as long experience has shown, will disappear spontaneously when the inflammation comes to an end. There seems to be no danger, however, in attempting to remove the stagnating fæcal masses by means of *enemata*, provided opium be given at the same time (as much as thirty drops twice or three times daily). When this plan of treatment has once been decided upon, it is to be carried out thoroughly by means of copious irrigations repeated every two or three hours, so as to secure if possible the breaking down and removal of the fæcal masses which are in part keeping up the peritonitis. I remember a desperate case of stercoraceous vomiting, meteorism, collapse, etc., in which defecation and relief were not obtained until after one hundred and twenty syringefuls had been perseveringly administered. According to the principles accepted nowadays respecting the admissibility of abstraction of blood in internal diseases, even the application of leeches, not to speak of the use of venesection, is to be condemned; mustard plasters and other rubefacients may, on the other hand, be used, but much benefit is not to be expected from them. If the peritonitis do not become general, and prove rapidly fatal, the chief indication will be to produce absorption of the exudation (pus), in order to prevent a further rupture into the peritoneal cavity, etc. The ice-bag should therefore now be replaced by *poultices*, which, according to my observation, are usually not employed sufficiently early in peritonitis. The time when the change is to be made depends so much upon which of the two plans of treatment gives the patient the greater comfort, that I shall not venture to decide this important question absolutely. *As a rule, I do not delay the tentative use of poultices later than during the second week.* Less is to be expected from the absorptive action of the tincture of iodine, etc. In view of the happy result obtained by Willard

Parker in opening a perityphlitic abscess through the abdominal wall, the question is to be seriously entertained, when the peritoneal exudation is encysted, whether an operation is not necessary to prevent the rupture of the pus internally. At all events, if fluctuation can be felt and the abscess points externally with reddening of the skin, the operation should at once be performed.

Another serious complication of ulcerations of the intestines is intestinal hemorrhage; but this is seldom so considerable as to require treatment. When treatment is necessary, on account of the quantity of blood lost and the exhaustion occasioned thereby, it is to be governed by the same rules as were laid down for the treatment of gastric hemorrhage. (See below, Intestinal Hemorrhages.) Hypodermic injections of ergotin, and the administration of alum-whey, and ice, are generally of some benefit when the bleeding comes from the upper parts of the intestines, while irrigations of ice-cold water, with or without the addition of astringents, are well suited to control hemorrhages arising from the colon. When the bleeding point is situated in the rectum, and is thus accessible to the direct application of styptics, the tampon, the application of lint saturated with the liq. ferri perchloridi and the actual cautery may be tried in succession.

If the general health of the patient has become impaired by the influence of the fundamental disease, by the loss of absorptive power on the part of the ulcerated intestine, by repeated hemorrhages, or by other debilitating causes, the first indication for treatment is to restore the strength by proper food, tonics, etc.—and this all the more because in the healing of intestinal, as in the healing of other ulcers, the more the general vigor of the individual improves the more confidently can a *restitutio ad integrum* be anticipated.

When healing has at last fortunately taken place, the subsequent disturbances occasioned by adhesions, narrowing of the intestine, etc., may still be a source of considerable annoyance to the patient, and may require treatment. At all events, the patient should be cautioned to observe prudence in diet for some time to come ; if chronic constipation occur, laxatives and irrigations should be regularly used, and, in strictures of the rectum, dilatation by bougies, etc. In particular cases the physician will be obliged to resort to a variety of remedial measures, the detailed indications for which must be left to his own judgment.

Neoplasms of the Intestinal Wall.

Neoplasms of the most diverse characters are found in the wall of the intestine: *adenomata*—particularly the papillary fibro-adenoma,—pure *fibromata*, *angiomata*, and *lipomata*,[1] in the form of tumors originating in the submucosa and sometimes projecting, as large polypi, into the intestinal cavity. Besides these, there occur also *lymphatic* new growths in cases of leukæmia, *myomata*,[2] (*melano-*) *sarcomata*—as a primary affection in the rectum, but usually developed secondarily in the intestinal wall in the form of numerous black specks and nodules[3]—and, finally, in rare instances, *cysts*, several cases having been observed by Rokitansky in which multilocular cysts filled with serum were partially imbedded in the intestinal wall.

The interest connected with all of these neoplasms is chiefly of a pathologico-anatomical character; clinically, they are of very subordinate importance, because they are incomparably less frequent than *carcinomata*. Like these, the tumors under consideration may give rise to symptoms of intestinal stenosis, to closure of the canals opening into the intestine (ductus choledochus, pancreaticus, etc.), or, under some circumstances, to hemorrhage. Some of them, like the lipomata, are of an entirely harmless character, and merely interfere to some extent with the normal play of the peristaltic movements; incidentally, however, they may probably also be the occasion of the occurrence of intussusceptions, in which the tumor is connected by its pedicle with the border of the orifice of the intussuscepted part (Rokitansky). But all such cases are clinical rarities, and always impossible to diagnosticate, unless the tumor happens to be situated low down in the rectum, so as to be accessible to direct examination.

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[1] *Virchow*, Die krankhaften Geschwülste. I. S. 382. Fig. 74. Abbildung eines Fettpolypen des Jejunums.

[2] *Virchow*, Die krankhaften Geschwülste. III. S. 133. Fig. 217. Abbildung eines submucosen Fibromyoms des Duodenums.

[3] Ibidem. II. S. 288. Abbildung metatastischer ulceröser Melanosarcome des Duodenums.

Intestinal cancer, on the contrary, presents a distinct and quite characteristic clinical picture of disease, which requires a more complete description.

Cancer of the Intestines.

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Pathological Anatomy.

As a general description has already been given of the structure of cancer in the article on Cancer of the Stomach, I shall merely remark here that in the intestines the epithelial nature of the neoplasm is strikingly marked. The epithelial cells of the Lieberkühnian follicles are found to be enlarged and elongated at their blind extremities, "the muscularis mucosæ is pierced in many places, the epithelial plugs project into the submucosa at the points of rupture, and, finally, in various places one can distinctly trace how a group of epithelial cells, still connected above with a gland, dips downwards directly into a rosary-shaped lymphatic vessel" (Waldeyer). The same writer thought that he found a very marked proliferation of the gland cells in the neighborhood of the solitary follicles of the rectum, the locality, therefore, where normally, the Lieberkühnian follicles, probably on account of the peculiar relations of the surrounding parts, appear to attain their greatest length, without being actually proliferated.

The same special characters which are presented by the cancerous disease in the stomach are found, also, in the intestines, except that here, as might be expected from the locality, the affection usually spreads in an *annular* form, and the lumen of the intestine becomes narrowed symmetrically, partly from the extension of the tumor inwards, partly from the cicatricial contraction which takes place in the matrix after the regressive metamorphosis of the cancerous masses has once begun. This process of disintegration may, however, after a while, make the intestine more permeable at the constricted point; or the obstruction to the passage of fæces may be relieved in another way, viz., the cancerous disease extends to an adjacent portion of intestine, firm adhesion takes place, subsequently a disintegration of the cancerous masses, and finally an abnormal communication is formed between the two portions of the intestine at the point of adhesion. The intestine above the constriction is dilated by an accumulation of fæces, while the portion below this point is contracted and empty.

In intestinal cancer, moreover, just as in cancer of the stomach, we find various secondary affections, such as inflammation of the mucous membrane in the vicinity of the neoplasm, thickening of the muscularis with separation of its bundles of fibres, prolapse of the affected part, on account of its increased weight, into the deeper abdominal regions, firm adhesion to the neighboring parts, ruptures into the peritoneum, extension of the neoplasm to the adjacent abdominal organs, fistulæ, hemorrhages, etc.

The *varieties* of cancer found in the intestines are the same as in the stomach: *scirrhus* and *medullary carcinoma*, according as the matrix or the cancer cells present the more advanced development. *Colloid cancer* is also of not infrequent occurrence. The *location* of the disease may be anywhere from the duodenum to the sphincter ani, but far more frequently the large intestine is affected, particularly at its flexures, in the sigmoid flexure (at both ends) and in the rectum.

According to the compilation of Köhler, out of thirty-four cases the cancer was situated twenty-two times—that is, in two-thirds of the cases—in the large intestine (the rectum excluded), in twelve in the small intestine (nine times in the duodenum).

Etiology.

The special frequency of cancer at the flexures—that is, at the narrower parts of the intestine—led Baillie to express the opinion that the main cause of the disease, and of its predilection for certain localities in the intestines, probably lies in the more marked development of the glands in the lower part of the large intestine, and in the greater susceptibility to injury by the passage of hard bodies at the point where the colon becomes contracted—that is, at the sigmoid flexure; these hard bodies, he concludes, "by their irritation may excite the disease of scirrhus in a part which was predisposed to it. What we have now said, however, is merely conjecture." Our knowledge of the etiology of intestinal cancer has not advanced a whit since the time of Baillie, except that the numerous cases reported since then, in which carcinoma of the intestines has apparently been produced by mechanical irritation, have rendered Baillie's view still more probable ; but we are still obliged to subscribe to the modest conclusion of his remarks. A recent case of Waldeyer's, in which, a year after an operation for ovarian dropsy, cancer developed in the portions of intestine which had become adherent to the pedicle (l. c., p. 14), shows very clearly the connection between mechanical inflammatory irritation and the development of intestinal cancer.

As in the stomach, so also in the intestines, cancer generally occurs as a *primary* disease, and is only to be regarded as *secondary*, either in those rare cases in which *metastatic* nodules are found in the intestinal wall in connection with a general dissemination of cancerous elements, or, when, as is more frequent, a cancer of the stomach, uterus, ovary, etc., extends *per contiguitatem* to the adjacent bowel.

Primary cancer of the intestines generally remains as an isolated tumor ; occasionally, however, the disease extends from this locality to various other organs, as was shown very instructively by a case of Bamberger's (l. c., Observation 1), in which a cancer of the cæcum gave rise, by means of embolism, to massive infiltrations in the serosa and liver.

Before the age of forty the disease has very rarely been met with ; still it has been observed even among children, Clare reporting a case of medullary cancer of the descending colon in a boy three years old. According to Tanchou's compilation, comprising more than nine thousand fatal cases of carcinoma, the relative frequency of intestinal cancer, as compared with that of other cancers in the body, is about one to twenty-five.

Symptomatology.

The first symptoms of intestinal cancer are of an indefinite character—fixed, dull pain at one point, distention of the abdomen, and irregular action of the bowels. Later in the course of the disease other symptoms occur, some of which are peculiar to cancer in general, while others are connected with the special locality occupied by the carcinoma. Under the first head is to be noticed particularly the *cachexia*, which is very marked when the disease runs a slow course, and occasions the well-known pallid appearance of the patient. Still the emaciation is not always pronounced, and is very apt to be absent when the case runs a rapid course with multiple cancerous deposits. Several very striking instances of this kind are reported by Bamberger.

One of these cases is so interesting and so typical of this form of the disease, that I give an abstract of it: A well-nourished woman, fifty-seven years of age, but looking fifteen years younger, was taken sick one month before her death with anorexia, vomiting, and pains in the epigastrium, followed, a fortnight afterwards, by a moderate degree of prostration. On examination nothing abnormal was detected anywhere, except in the right side of the abdomen, where, on deep pressure, a *non-painful* tumor, hardly as large as a pigeon's egg, could be felt, which increased within four days to the size of a fist. The liver and spleen were of normal size. Four days later, after the bowels had been moved by a cathartic, the liver appeared enormously swollen, and was uneven, hard, and painless to the touch. The feebleness and meteorism increased, and death ensued two days afterwards. Autopsy: Medullary cancer of the cæcum and cancerous adhesions of several convolutions to each other; multiple cancer of the peritoneum, and especially, also, of the mesentery, and complete infiltration of the considerably enlarged liver, with numerous carcinomatous nodules, which, as the thrombi in the portal vessels proved, had been of embolic origin.

Abdominal *pain* is also a very uncertain symptom of cancer, as is shown by the case above quoted. In intestinal cancer, particularly, the presence of pain is the less significant, from the fact that here the symptom is accompanied by colicky pains, and cannot, therefore, be so directly traced to the local disease.

The most important member of the complex of symptoms, and the one which really first gives a definite direction to the diagnosis, is the appearance of a palpable *tumor*. This is hard, somewhat uneven, dully-tympanitic on percussion, and pulsative, when situated over the large abdominal vessels. In consequence of the massive accumulation of fæces above the tumor, the latter appears during life much larger than corresponds to the actual size of the carcinoma; hence the existence and dimensions of the carcinomatous tumor can be positively ascertained only by the thorough use of cathartics and irrigations. Of special diagnostic importance is the determination of the *position* of the tumor. Sometimes the spot occupied by the tumor corresponds to the normal situation of the affected portion of intestine. This is the case when the carcinoma develops in the cæcum, ascending colon, etc.—in short, in those portions of the intestine which are incapable of extensive movements on account of their close attachments to neighboring parts. In cancer of the small intestine and transverse colon, on the other hand, the mobility is almost unlimited so long as no adhesions have taken place to the fixed organs of the abdomen. Such mobile tumors sink of their own weight into the deeper abdominal regions, are displaceable, and change their position according to the movements of the intestine—in fact, *a frequent change in the position of the tumor, and in the distinctness with which it can be felt*, in consequence of the shifting of the intestinal convolutions over each other, a temporary accumulation of fæces, etc.—*is to a certain degree characteristic of intestinal cancer.*

As a mechanical result of the usually annular development of cancer in the intestine, the fæces accumulate for some distance above the constricted part, irritate the mucous membrane, and may thus excite attacks of diarrhœa alternating with constipation. More and more the *symptoms of intestinal stenosis* now advance into the foreground—distention of the abdomen, colicky pains, vomiting, violent peristaltic, or, at last, anti-peristaltic movements of the intestine with stercoraceous vomiting, the passage of small, thin flattened lumps like the fæces of sheep, moulded by the stenosed lumen of the intestine, and finally peritonitis.

The character of the stools is also of great diagnostic importance, and is in some cases directly pathognomonic. Besides the above-mentioned alterations in the form of the discharged fæces—alterations, however, which are not diagnostic of intestinal stenosis if fæces of normal shape are discharged at intervals—the stools are also found to contain blood, a gangrenous stinking fluid, and pus. The lower down in the large intestine the cancer is situated, the more unmistakable and the less altered are these abnormal ingredients of the dejections. In rare cases, detached pieces of cancer may also be met with; Wunderlich found in the stool of one of his patients "a cancerous mass as large as a walnut, the passage of which had been accompanied by severe hemorrhage and pains like those of labor."

As the disease progresses and disintegration of the neoplasm takes place, the above symptoms become aggravated, and finally rupture of the intestine takes place into the peritoneal cavity or an adjacent portion of the intestine, or a fæcal fistula is formed by a rupture into the bladder, vagina, or, in rare cases, through the external integuments. The establishment of such a fistula naturally gives relief for a time, by affording a new outlet for the fæces; but this temporary improvement is the more disappointing on account of the unfortunate results which ensue. Aside from the danger of a general peritonitis, which almost always proves fatal within a few days, and aside from the discomforts of an external fæcal fistula, a rupture into the bladder or vagina is followed by the formation of a disgusting cloaca, which is a source of extreme annoyance to the patient. The fæces now pass off through the urethra or vagina, along with the urine or secretions of the sexual organs and the bloody and gangrenous discharges from the cancerous surface. Flatus also escapes through the urethra; in Formey's noted case, for example, this symptom was the first indication that the cancer of the rectum had perforated into the bladder.

The final result is death, from the gradual effects of general exhaustion, unless its occurrence is hastened by a rupture of the intestine, by the supervention of pyæmia, by venous thrombosis, and embolism, or by some similar rapidly fatal catastrophe. In certain cases the symptoms of the intestinal cancer are overshadowed by those belonging to the secondary development of the disease, particularly the symptoms of cancer of the liver, with considerable enlargement of the organ, and by those of peritoneal cancer with ascites.

Certain modifications of the above picture of the disease are sometimes occasioned by the locality of the tumor. Since quite an important part of the symptoms depends entirely upon the mechanical effects produced by the stenosing neoplasm, in the parts situated above, it is obvious that, in this respect, cancer of the duodenum must differ essentially from cancer in other portions of the intestines, because, in duodenal cancer, these secondary effects are exerted upon the stomach. Accordingly, during the course of this form of the disease, we find vomiting with or without blood, cardialgia, gastric derangements in general, finally dilatation of the stomach, absolute absence of stools with retraction of the abdomen—in short, the complete picture of pyloric stenosis, so that a differential diagnosis between the two diseases is impossible. If a tumor can be felt, it will be found on the right side between the umbilicus and the border of the right ribs, tuberculated, resistant, and generally entirely immovable. At all events, if movable to any considerable degree, it must be situated in the superior, horizontal portion of the duodenum, because this is the only portion normally capable of much locomotion.

On the other hand, carcinoma at the outlet of the intestines, in the lowermost part of the *rectum*, will give rise to symptoms of a different character, partly because this portion of the intestines has a special function—the discharge of the fæces—and therefore suffers in a special manner, and partly because the cancerous lesions can be directly felt or even seen.

The patient experiences burning pains in the rectum during defecation, or on long standing or sitting, and these pains radiate into the surrounding parts, the sacrum, the genitalia, along the course of the ischiatic nerve, etc. Sometimes actual tenesmus is present, a feeling as if there were something still left in the rectum after the bowels have moved. The evacuations are in most cases difficult and painful, and for this reason the patient postpones the defecation as long as possible, as a natural result of which the fæces accumulate in still larger quantities above the constriction, excite irritation and catarrhal inflammation of the rectal mucous membrane, and occasionally the formation of ulcers. Moreover, the retained fæces, as well as the neoplasm itself, interfere with the return of the venous blood, and thus varicose swellings of the rectal veins, or *hemorrhoids*, are produced, which, on their part, add a new obstruction to the evacuation of the bowels and increase the already terrible sufferings of the patient. When a catarrh of the rectal mucous membrane has developed under these conditions, the constipation is interrupted by attacks of temporary diarrhœa, and, finally, if atony of the intestinal muscles, and particularly if paralysis of the sphincter ani, supervene, a continuous flow of thin brown sanious fluid streams from the anus, and excoriates the surrounding parts. Besides these derangements affecting the rectum itself, others also occur on the part of the *adjacent organs*, particularly the bladder, in the form of strangury, retention of urine, and enuresis. Compression of the orifices of the ureters, resulting in hydronephrosis, may also be produced by the extension of the neoplasm to the posterior wall of the bladder, and in the later stages the recto-vesical wall may break down, and an abnormal communication be thus established between the two viscera, with the results already described. In the same way recto-vaginal fistulæ may arise, as well as rectal fistulæ with destruction of the anus.

The *examination* by the rectum, which should never be neglected in cases of intestinal and particularly of rectal cancer, discloses rough, hard nodes, over which the mucous membrane is still movable ; later in the disease, the finger encounters a close ring of nodes, through which it is impossible to pass, or an ulcerated surface which soils the finger with blood, ichor, etc. Certainty, however, in regard to the *cancerous* nature of the ulcer requires the use of a rectal speculum or endoscope ; but unfortunately their introduction causes extreme pain. In addition to the presence of carcinomatous nodes, the manual examination sometimes reveals complete immobility of the rectum and induration of the surrounding tissue. Additional information may be gained by examining per vaginam, and, when the cancer is situated high up in the rectum, by the introduction of a rectal bougie.

In rectal cancer, also, death may result, simply from the constipation and the finally ensuing ileus. In some cases, the constipation may be enormously prolonged; thus Fallot reports a case of rectal cancer which terminated with a constipation of sixty-one days' duration. The fatal result may be induced or at least accelerated also by the various complications mentioned above.

Diagnosis.

The *diagnosis of intestinal cancer*, if we except the instances in which a rectal carcinoma can be detected by an examination per anum, almost always presents difficulties. In this remark I exclude entirely the cases in which there are no objective symptoms, and the patient merely suffers from dull abdominal pains, with tympanites, an irregular action of the bowels and symptoms of cachexia; under such circumstances a diagnosis is altogether out of the question. But even when a *tumor* can be distinctly felt, the greatest caution is necessary in making a diagnosis, because it is only too easy to mistake an intestinal cancer for other abdominal tumors. Not to mention the corset-liver and tumors of the spleen, which, when carefully examined, are not likely to be readily confounded with intestinal cancer, mistake may arise from a renal cancer, a tumor of the mesenteric glands, an encysted peritoneal exudation, or a chronic inflammatory infiltration of circumscribed portions of the intestine after typhlitis and perityphlitis. In the case of the first two of these tumors, it must be borne in mind that they develop *behind* the intestines, and are not directly in contact with the abdominal wall until they have attained considerable size, while the other abdominal tumors mentioned can usually be distinguished from intestinal cancer by the history of the case and by the considerable dimensions of the infiltration and exudation (extending even into the parts surrounding the infiltrated intestine). I would particularly call attention once more to the *variableness* in the position and palpability of the tumor as characteristic of intestinal cancer, such changes being less noticeable in all the other tumors mentioned. But probably the most frequent question to be decided, is whether a solid tumor felt in the course of the intestines is an intestinal cancer or a purely fæcal tumor, or whether a fæcal accumulation has gradually taken place above the site of an intestinal carcinoma. The doubt in such cases can generally be cleared up by the persistent use of irrigations and cathartics. The appearance of unmistakable symptoms of intestinal stenosis in connection with the above-mentioned characteristic changes in the stools will add materially to the evidence from physical examination, and the diagnosis becomes absolutely certain if cancerous elements can be detected in the dejections, while a considerable degree of certainty is attained if the presence of a tumor is associated with the discharge of flattened fæces, blood, etc., although, even under these circumstances, errors in diagnosis are still possible.

Thus, Wunderlich (l. c., 227) gives an account of a case in which the apparent symptoms of intestinal cancer—nodular tumor, severe lancinating pains, and passage of flattened fæces—were all ultimately found to have been produced by an aneurism of the iliac artery.

When we have become satisfied, from the history of the case, the advanced age of the patient, the enlargement of the peripheral glands, the cachexia, the character of the stools, the intestinal stenosis, and from other results of examination, that the case is one of cancer of the intestines, the *next question* to be decided is, *which portion of the intestinal canal is occupied by the neoplasm.*

Cancer of the duodenum *cannot* be absolutely distinguished from pyloric carcinoma. The former, however, may be suspected if the disease be accompanied by obstinate *jaundice*, this symptom occurring in pyloric cancer merely as an accidental complication, while in carcinoma of the duodenum it is generally a necessary result of the locality affected. Moreover, if a discharge of blood take place *only* in a downward direction, such an occurrence will point, in a doubtful case, to a duodenal rather than a pyloric cancer, because in the latter the blood is usually discharged by vomiting. Furthermore, the duodenal cancer is liable, on account of its position, to be confounded with an enlarged gall-bladder, or with the head of a healthy pancreas which has become accessible to palpation (see above, Cancer of the Stomach).

Cancerous disease in the *small intestine*, if the affected portion of intestine be not fixed by adhesions, is characterized by the great active and passive mobility of the tumor. When the latter attains considerable dimensions, it drags down with it the affected convolutions into the lower regions of the abdomen.

The same is true of cancer located in the movable *transverse colon.* After the prolapse of the neoplasm has taken place, the case is most apt to be mistaken for one of pyloric cancer, and the diagnosis must then turn upon the presence or absence of a gastrectasia.

Cancer of the *ascending colon* or *cæcum* may also be confounded with a prolapsed pyloric cancer, when the former, as sometimes happens, is displaced far over towards the median line. Thus, I recently dissected a cancer of the cæcum which during life had been constantly felt, for a long time, in the right parasternal line, two inches below the border of the ribs, as a nodular, tender tumor, about four ctm. in diameter. The distinction between the carcinomata of the transverse colon and the carcinomata of the ascending colon, descending colon, and sigmoid flexure, consists in the slight or complete immobility of the latter tumors, the most movable of them being the carcinoma of the sigmoid flexure, since this portion of the bowel has a long mesentery, and is therefore capable of undergoing displacements.

When the stenosis is situated exactly at the ileo-cæcal valve, trial may be made of a diagnostic expedient devised by Bourdon, and resorted to by him in one case. The fæcal tumor, which arises from the accumulation of fæces above the thickened valve in consequence of the inability of the small intestine to overcome the obstruction, can be made to disappear by pressing upon the abdomen and thus forcing the fæcal masses through the stenosis.

Cancer of the rectum is most conveniently situated for purposes of diagnosis, because it is usually within reach of the finger. But whether the neoplasm, projecting into the cavity of the rectum, be of a benignant or of a malignant character, it is difficult to determine from a simple examination, unless the case be one of pedunculated polypus or of well-marked cancerous ulceration. The decision must be made from the history of the case, the further course of the disease, and from a microscopical examination of an excised portion of the neoplasm. The differential diagnosis lies very generally between cancer and hemorrhoidal tumors. The latter have a smooth surface, a firm but elastic consistence, and are not tender when pressed with the tip of the finger—characteristics which sufficiently distinguish them from rectal cancer, in which, moreover, besides the blood in the stools, there is usually an ichorous discharge flowing from the anus.

One method of *ascertaining approximately the height at which an intestinal cancer is situated* consists in throwing injections into the intestine, after removing the fæces from the parts below the cancerous stenosis. The less the amount of fluid that can be injected, and the more rapidly the water is discharged, the lower the site of the obstruction. Still more definite conclusions can be reached if, after the injection has been given, the abdomen be percussed, to determine the height to which the water can be forced into the intestine. On its escape, the water should be carefully examined for blood and cancerous particles. Of course, this diagnostic expedient has usually only a subordinate value.

Prognosis.

The prognosis in intestinal cancer, as in other cancers in which extirpation is impossible, is absolutely *bad*. To be sure, it cannot be denied that the carcinoma occasionally manifests a certain tendency to cure. A case of this kind has been described by Rokitansky, in which an annular medullary cancer of the sigmoid flexure, not quite four inches in length, had become cicatrized for the space of three inches and a half, and was composed, to this extent, of a stratum of connective tissue free from cancer corpuscles. Still, such cases are extraordinarily rare, and show, at most, that the partial healing which had occurred might in the course of time have terminated in a complete cure. The best prognosis is afforded by the rectal cancers when they can be completely extirpated, and the worst by those cancers which do not remain confined to the intestine, but extend rapidly, and run an acute course, as in Bamberger's case cited above. This form of cancerous disease, which presents the features of an infective process, may prove fatal within a few weeks, while other intestinal cancers may apparently continue for years without inducing a fatal result.

Thus, a case of cæcal cancer was observed at the Erlangen Polyclinic, which, as was shown by the case-book, had been diagnosticated three years before as a carcinoma ventriculi et coli (?). The patient himself had for two years and a half felt a tumor in the abdomen, which had at times been very painful.

Treatment.

Internal cancer, being an incurable disease produced by unknown causes, can receive only a *symptomatic* treatment. The main indications are *the regulation of the bowels* by means of enemata and mild cathartics, in order to obviate the serious results of constipation, the *selection of food which is easily digestible and produces as little fæces as possible, and the use, when necessary, of narcotics,* to relieve the distressing pains. In cancers of the rectum, when an operation is impracticable, the parts should be repeatedly washed out with water or some disinfecting fluid, and now and then a large bougie may be introduced through the stricture, not so much for the purpose of dilatation, which is always dangerous from the risk of producing perforation, as to provide an outlet for the gases and fæcal masses which have accumulated above the site of the cancer. In order to facilitate the escape of the fæces, the attempt may also be made to soften them by means of water injections through a tube introduced high up. If this measure do not succeed, and if the lumen of the intestine be practically occluded by the neoplasm, it would be proper to employ artificial dilatation to prevent the rupture of the retained fæces into the surrounding parts.

Dieffenbach reports a case in which it was necessary to make deep incisions into both buttocks to allow the escape of an immense quantity of fæces which had accumulated there as a result of perforation of the intestine and had given rise to dangerous symptoms.

Hemorrhages are to be checked by the usual remedies, strangulated hemorrhoids should be replaced, etc. If the measures above indicated fail to relieve distressing results of the constipation and dangerous symptoms arise from this cause, the establishment of an artificial anus above the constricted point is indicated (Callisen and Amussat). Recently, Curling has recommended colotomy, even as a palliative measure, in cases of rectal cancer unattended by constipation, on account of the relief from pain and the restoration of strength which are said to follow the operation.

In regard, finally, to the indications and the methods for the complete extirpation of rectal cancer, I must refer the reader to the manuals of surgery.

Intestinal Hemorrhage.

Comp. the literature of gastric hemorrhage.—Special articles on intestinal hemorrhage: *Jos. Frank*, Prax. medic. praec. Pars III. Vol. II. Sect. I. p. 698, with a full account of the older literature.—*Albers*, Die Darmgeschwüre. 1831. S. 168.—*Dassen*, Ueber die Quelle des beim Morbus niger. etc. Pract. Tijdschr. 1838. Schmidt's Jahrb. Bd. XXII. S. 172.—*Graves*, Clinical lectures. 1838.—*Jacksch*, Practische Mittheilungen, etc. Prager Vierteljahrschrift. II. 1. 1845.—*Finger*, Blutungen beim Typh. exanthem. etc. Prager Vierteljahrschrift. 1849. VI. Jahrg. 3. S. 28.—*Kniesling*, Ileocolotyphus mit tödtl. Darmblutung, etc. Allg. Bemerkg. Deutsche Klinik. 1850. No. 14. S. 148.—*L. Traube*, Hämorrhagie beim Typhus. Charitéannalen. 1850. Gesammelte Beiträge, etc. II. S. 26.—*Schweich*, Notiz, etc. Deutsche Klinik. 1851. S. 249.—*Virchow*, Gesammelte Abhandlungen, etc. S. 420. 438. 451.—*Albers*, Bericht über 84 Leichenöffnungen, etc, etc. Deutsche Klinik. 1851. No. 4.—*Canstatt*, Specielle Pathologie u. Therapie. 1856. Bd. III. S. 402.—*Beckmann*, Embolie der A. mes. sup. Virchow's Archiv. XIII. S. 501. 1858.—*Finger*, Ueber spontane (Darm-) Blutungen. Oesterreichische Zeitschrift f. pract. Heilk. V. Ref. Schmidt's Jahrb. Bd. 107. S. 301. 1859.—*Geist*, Klinik der Greisenkrankheiten. 1860. II. S. 145.—*Cohn*, Klinik der embolischen Gefässkrankheiten. 1860. S. 546. ff.—*Frerichs*, Klinik der Leberkrankheiten. I. 1861. S. 357.—*Oppolzer*, Embolie der A. mesent. sup. mit Enterorrhagie. Allg. Wiener med. Zeitschr. VII. 1862. Ref. Schmidt's Jahrb. 117 S. 239.—*Gerhardt*, Embolie der A. mesentericae. Würzb. medic. Zeitschr. Bd. 4. S. 141. 1862.—*Kussmaul*, Zur Diagnose der Embolie der A. mesent. ibid. Bd. 5. S. 210. ff. 1864.—*Vallin*, Schmidt's J.-B. 1864.—*Griesinger*, Infectionskrankheiten. 1864.—*Moos*, Beitrag zur Casuistik der embolischen Gefässkrankheiten. Virchow's Archiv. Bd. XLI. S. 58. 1867.—*Henoch*, Ueber den Zusammenhang von Purpura und Intestinalstörungen. Berliner klin. Wochenschrift. 1868. No. 50. S. 517.—*Grainger Stewart*, On hemorrhage of waxy, etc. Brit. and For. Med. Chir. Review. 1868.—*Hegar*, Embolie der A. mesaraica inferior. Virchow's Archiv. Bd. 48. S. 332. 1869.—*Markwald*, 3 Fälle von Darmblutung beim Typhus (präs. *L. Traube*). Diss. Berol. 1869.—*Vallin*, Observ. d'haemorrhagie intestinale. Union med. 1869. Canstatt's J.-B. II. 147.—*Ponfick*, Zur Casuistik der Embolie der A. mesent. sup. Virchow's Archiv. Bd. L. 623. 1870.—*O. Bayer*, Tödtliche Darmblutung in Folge transitorischer Hyperämie bei Erysipelas faciei. Archiv für Heilkunde. 1870. S. 399.—*Wunderlich, V. C. A. jun.*, Ueber Darmblutung beim Typhus abdomin. etc. Archiv für Heilkunde. 1872. S. 481. ff.—*Scheby-Buch*, Gelenkaffectionen, Darmblutung, etc. Deutsches Archiv für klin. Medic. Bd.

XIV. S. 460. ff. 1874.—*J. Faber*, Die Embolie der A. mesenterica sup. Diss. Inaugur. Erlangen. 1875.—*Litten*, Ueber die Folgen des Verschlusses der A. mesar. sup. Virchow's Archiv. Band 63. S. 289. 1875.

The same general principles, which we have found to govern the occurrence and course of gastric hemorrhages, apply also in almost every particular to the bleedings which take place in the intestines. In order to avoid, therefore, a mere repetition of our previous remarks under the former head, we shall mention only briefly the points common to both diseases, and shall dwell more at length upon the points in the pathogenesis, symptomatology, etc., which are characteristic of intestinal hemorrhage, occurring independently of hemorrhage from the stomach.

Etiology and Pathogenesis.

As in gastric, so also in intestinal hemorrhage, the immediate cause of the bleeding is an abnormal force of the blood-pressure, or, still more frequently, a morbid condition of the wall of the vessel. Thus, on the one hand, we have intestinal and gastric hemorrhages occasioned particularly by stases in the area of the portal circulation, as in liver diseases, obstructions of the vena portæ, and diseases of the lungs, heart, and great vessels; on the other hand, intestinal hemorrhage may arise from diseases and hurtful influences which involve lesions in the structure and continuity of the walls of the vessels. Under this head may be included mechanical or chemical injuries to the intestine (foreign bodies, wounds, poisons), aneurisms projecting into the lumen of the bowel, and amyloid degeneration of the wall of the vessel (Stewart), occurring especially in previously syphilitic individuals. A very interesting form of transient hyperæmia of the intestine with enterorrhagia was observed by Bayer in a case of facial erysipelas, the disappearance of which was followed by a copious hemorrhage into the small intestine. Similar transitory hemorrhages in the intestines ensue after severe and extensive burns of the skin of the abdomen, as well as in anomalies of menstruation, in which a bleeding from the bowels sometimes takes the place of the normal menstruation; upon this point the remarks in the article on Gastric Hemorrhage will apply here also. Intestinal hemorrhages occur, moreover, in the course of diseases which are in general accompanied by bleeding, viz., hæmophilia, the scorbutic affections, particularly *purpura hæmorrhagica, with and without joint affections*, yellow fever, malignant jaundice, etc. In this connection it should be borne in mind also that severe *intermittent fever* is sometimes attended by intestinal hemorrhages. Frerichs, who has observed several instances of profuse intermittent bleedings from the bowels, ascribes such cases to a plugging of the capillaries of the liver with flakes of pigment, which induce hemorrhage by damming up the blood in the radicles of the vena portæ.

While in the cases mentioned thus far the intestinal bleeding forms but a single link in the chain of hemorrhages, which may arise in a great variety of organs as the effect of a common cause —the intestinal hemorrhage being, therefore, merely a more or less accidental result of the fundamental disease—in other morbid conditions the intestinal vessels are affected exclusively. Such specific hemorrhages may, in the first place, be produced by intense *enteritis*, in which the special violence of the inflammation probably occasions a very marked degree of retardation of the blood-current in the capillaries, and a copious extravasation of red blood corpuscles upon the free surface of the intestinal wall. This extravasation, *ceteris paribus*, will take place the more readily the greater the morbid changes in the walls of the vessels before the occurrence of hemorrhage ; hence the frequency of this symptom in the enteritis of old age. The form of inflammation most frequently accompanied by intestinal hemorrhage, is the *diphtheritic*, and in this remark we include both the infective form of intestinal diphtheria and that which occurs during the course of severe infective diseases ; in fact, Bamberger declares that dysentery is the most frequent of all the causes of intestinal bleeding.

The next most frequent cause is *typhoid fever*, in which the hemorrhage may proceed either from the capillaries without rupture, or from the large vessels which have been eroded in the bases of the typhoid ulcers.

A striking example of the first form of bleeding is reported by Markwald. The case was one of typhoid fever in which a profuse intestinal hemorrhage had occurred; at the autopsy only small ulcerations were found, and on injecting the superior mesenteric artery with a colored fluid the finest vessels of the intestinal mucous membrane were filled without the escape of even a drop of the fluid from the site of the ulcers.

Besides the typhoid *ulcerations*, all the other forms of ulcer previously described—the *catarrhal*, the *peptic*, and the *tuberculous*—not unfrequently give rise to intestinal hemorrhage. Duodenal ulceration, in more than one-fourth of the cases, is attended by hemorrhages, which are generally of a profuse character, the blood escaping by vomiting alone, or only by stool, or in both ways at the same time.

Neoplasms of the intestine, especially cancer, are also occasionally attended by enterorrhagia. The tendency to hemorrhage is particularly marked in cancer of the rectum, because here, in addition to the bleedings which result from the disintegration of the neoplasm, hemorrhages may also occur from the stases in the inferior veins produced by the pressure of the carcinoma or impacted fæces. In such cases the visible evidence of a considerable obstruction to the circulation is presented by the appearance of *hemorrhoids* at the outlet of the rectum.

Of a similar origin are the bleedings from the rectal veins, which occur in *chronic constipation*, or during the course of *pregnancy*, from the compression of the veins by the gravid uterus. When *hemorrhoids*[1] have once arisen from this or some other cause, bleeding may be produced, either by a rupture of the tensely distended and thinned walls of the hemorrhoidal veins, or by inflammation and erosion of the tumors.

Another form of intestinal hemorrhage, originating in venous congestion, is that which occurs in *intussusception* of the intestine, owing to the fact that the mesentery is dragged into the sheath along with the invaginated part, and its vessels thereby undergo a considerable amount of compression.

Finally, intestinal hemorrhage has been repeatedly traced to an *embolism of the mesenteric arteries*. The explanation of such cases presents serious difficulties.

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[1] The subject of hemorrhoidal diseases will be fully treated in another portion of this work.

In his classical work on the embolic process, Cohnheim, as is well known, has shown conclusively that in the formation of the infarction it is the backward current from the veins that distends the vessels of the embolized district, and thus ultimately gives rise to hemorrhage. This result happens, however, only when the embolism occurs in what are known as the "terminal arteries," no such effect being produced when the occluded artery, before breaking up into capillaries, anastomoses with another artery. But the intestine is the place, of all others in the body, where the arterial anastomoses are most numerous and most perfectly arranged, and therefore it seems impossible to explain the occurrence of infarction after embolism of the superior mesenteric artery so long as the circulation can continue through the anastomotic connections of this artery with the cœliac artery by means of the pancreatico-duodenalis, and with the inferior mesenteric, by means of the middle cœliac. On the other hand, the occurrence of hemorrhagic infarction of the intestine, after embolism of the superior mesenteric artery, has been shown beyond question in numerous instances. In order to reconcile this discrepancy, Cohnheim has supposed that in all such cases occlusion occurs not merely in a large branch of the superior mesenteric artery itself, but also in the arterial anastomoses, which empty into the occluded artery between the original embolus and the capillaries of the intestinal wall, the secondary occlusion resulting from particles which have become separated from the primary embolus and have been swept off into the anastomosing vessels.

This explanation, which was based upon the results of two autopsies made by Ponfick, presupposed, however, that the secondary plugs had generally been overlooked at post-mortem examinations; but it is difficult to believe that this could have been the case. Faber, who failed to find any secondary plugs, has suggested another explanation in place of Cohnheim's, viz., that, notwithstanding the patency of the collateral vessels, the portal vein produces a retrograde pressure, which, in the absence of any counterbalancing pressure in the superior mesenteric vein, occasions an engorgement of the district supplied by the latter vessel.

M. Litten has recently made a number of experiments, which afford a satisfactory solution of the problem. These experiments show that when the superior mesenteric artery is ligated, the circulation in the vascular district supplied by the artery is never restored through the collateral circulation. On ligating a central portion of the artery, and dividing the vessel at its periphery, no blood escaped at any time up to the death of the animal. In every case, however, the ligation of the superior mesenteric artery was followed by an infarction of the portion of intestine supplied by the vessel. The first hemorrhages occurred from ten to twelve hours after the application of the ligature, and *were occasioned by the establishment of a retrograde venous current*[1] in connection with changes of nutrition in the anæmic walls of the vessels—changes which occur as early as from two hours to two hours and a half after the operation. Functionally, therefore, if not anatomically, the superior mesenteric behaves as a terminal artery, and for this reason Litten has appropriately given it the name of a "functional" terminal artery.

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[1] Litten has observed this retrograde venous current under the microscope, and has shown, by experiments with injections into the vessels, that the resistance in the ligated vascular district is so great as to prevent a *rapid* restoration of the circulation through the normal blood pressure on the part of the arteries.

On the other hand, the *inferior mesenteric* artery, on account of its numerous anastomoses, has *no* claim to this designation. When its embolization does result in hemorrhage (but without necrosis and œdema in the affected portion of intestine), the event is, according to Litten, to be explained by the fact that sufficient time elapses before the restoration of the circulation to allow the occurrence of such a destruction of the vessels as will permit the escape of the constituents of the blood when the arterial current becomes re-established. Such bleedings have, therefore, no connection with a retrograde current in the veins.

While gastric hemorrhages occur more frequently in women, the reverse is the case in regard to hemorrhages from the intestine. The absolute frequency of intestinal hemorrhages can scarcely be determined, because they occur merely as a symptom of various affections, and consequently vary in frequency with the frequency and intensity of the latter. Of more practical importance is the question, what particular diseases are found by experience to predispose most frequently to the occurrence of this event. Bamberger has drawn up the following scale, showing the direct order of frequency: dysentery, typhus, cancer (of the large intestine), mechanical hemorrhage, poisoning, and foreign bodies, tuberculous, follicular, catarrhal ulcers, and inflammation of the mucous membrane, the round duodenal ulcer, aneurisms, and finally vicarious hemorrhages.

Pathological Anatomy.

In individuals dying from profuse intestinal hemorrhage the body presents the signs of general loss of blood. On opening the abdominal cavity, the mesentery, in some instances, appears engorged with blood, especially in cases of congestive hemorrhage, and the intestine, on section, is found to be intensely hyperæmic; at other times, however, the mucous membrane is very pale, in consequence of the copious hemorrhage which has occurred during life. In still other cases the mucous membrane has a dark speckled appearance, which is produced partly by ecchymoses, and partly by effused blood, which may be pressed out of the orifices of the follicles.

The cavity of the intestine is filled with dark, blackish-brown, tarry clots of blood, but sometimes the blood is red in color and of a more fluid consistence. After removing these clots it is generally possible to discover the cause of the hemorrhage, either in the form of an eroded vessel lying in the base of an ulcer, an invagination, an embolism of the superior mesenteric artery, an aneurism, cancer, etc., or in the form of an intense enteritis, an amyloid degeneration of the intestine or diffuse congestion in the ramifications of the portal vein. In such cases of capillary hemorrhage the explanation of the bleeding will not be found until inquiry is made into the pathologico-anatomical condition of the liver, heart, etc.

Symptomatology and Diagnosis.

The symptoms which precede intestinal hemorrhage will naturally vary according to the nature of the disease which gives rise to the bleeding: congestive intermittent fever, typhoid fever, cirrhosis of the liver, purpura, etc.

When the intestinal hemorrhage is profuse, its occurrence is manifested by the general signs of internal hemorrhage: weakness of the pulse, pallor of the skin and mucous membranes, giddiness, ringing in the ears, glimmering before the eyes, a feeling of weakness and faintness; at the same time the patient complains of pain in the abdomen, and there is said to occur, in some cases, a sensation as if "a warm fluid were flowing into the abdomen." These symptoms may be immediately followed by death (internal hemorrhage), without any discharge of blood externally, owing either to the rapidity and enormous quantity of the extravasation or to a paralysis of the peristaltic movements. Usually, however, an inclination to evacuate the bowels is felt, and the patient has one or more discharges, consisting of more or less pure blood in the form of tarry masses, and sometimes containing fæces. In rare cases the extravasated blood may be *bright red*, but probably only when it has been discharged in very large quantities and has been rapidly hurried through the intestines, or when it has come from the lowermost portions of the intestinal canal, because, if the blood have been long delayed in the large intestine, the acidity of the contents of the latter is sufficient to produce a transformation of the hæmoglobin into hæmatin. When the hemorrhage occurs in the duodenum, the blood may also pass into the stomach, and there undergo the conversion into hæmatin.

Should the bleeding continue, or should repeated hemorrhages occur in quick succession, the anæmic symptoms become more and more threatening; the pulse disappears, the cheeks and eyes become sunken, a swoon relaxes the rest of the body, the temperature steadily falls, and the extreme collapse finally results in death. If the bleeding be arrested and the patient recover from this apparently hopeless condition, the bloody stools are gradually replaced by normal dejections, but the convalescence is protracted, and even when there is no affection of the kidneys, dropsy may still occur as a result of anæmia, and may ultimately prove fatal through an œdema of the glottis. Relapses of the bleeding are very common, and in some cases may be expected with certainty, as is evident from a glance at the etiology of intestinal hemorrhages.

The preceding symptoms belong, however, only to the more severe hemorrhages; the minor bleedings frequently escape observation, because they produce none of these signs of collapse, and are, therefore, easily overlooked. The hemorrhoidal bleedings, on the contrary, even afford the patient relief from the burden of an engorged portal system, at least immediately after the loss of blood.

Usually there is no difficulty in deciding at once and with certainty, *whether the discharged masses contain blood or not.* When large quantities of red blood escape, it is scarcely supposable that a physician can be deceived; but when the fæces are tinged with a blackish-green color by bile, or are blackened by the use of iron, they may be mistaken for the tarry substances, discharged in all cases of intestinal bleeding, in which the blood has delayed for some time in the bowel. In such cases all doubt will be removed by the microscope or by a spectroscopic examination for hæmatin.[1]

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[1] Hoppe-Seyler, II. Auf. S. 367.

Much more difficult than this question, whether the stool contains blood, is the question, *whether the blood comes from the intestine at all, and, if so, from what particular portion.* If the blood adheres as a reddish coating to the fæcal balls without any blood being found in their interior, the blood can come from no other source than the large intestine, and then the first diagnostic indication is to examine the abdomen along the course of the colon, and to make a manual or instrumental exploration of the rectum. If, on the other hand, large quantities of black, or even red blood are discharged, it is impossible, from a mere inspection of the fæces, to decide whether the source of the bleeding lies in the small or large intestine. Ordinarily the rule holds good, that the greater the distance which the blood has to travel before its final discharge, the more marked the changes it undergoes ; and yet we sometimes find in the hemorrhages of typhoid fever that the blood extravasated in the ileum appears of a red color in the stools. *Percussion* is of but little service in determining the particular location of the bleeding, except in the very rare cases in which an extensive dulness over the intestines can be detected which continues to increase in size. By this means we may also occasionally be enabled to determine whether the discharged blood comes from the upper portion of the intestines or from the stomach—a point which could not be determined by a mere inspection of the bloody stools. In most cases, however, the symptoms which precede and accompany the intestinal hemorrhage will indicate its situation with an approximate degree of certainty. Thus, in typhoid fever, we may regard the ileum as the probable source of the bleeding ; in dysentery, the large intestine, etc.

Usually, therefore, the doubt as to the locality of the hemorrhage will be solved *when we have settled, what is by far the most important point in the diagnosis and treatment, viz., the particular fundamental disease to which the intestinal hemorrhage is due.* This is of course not the place to discuss the symptomatology of these different fundamental diseases, cirrhosis of the liver, typhoid fever, etc.; still it may be of service to give certain general diagnostic points, which will enable us to make a rapid survey of the case.

When the antecedents of the patient are unknown, and the bloody discharges present none of the characteristic appearances of *dysenteric stools*, the first indication, after the general condition, the state of the pulse, the temperature of the skin, etc., have been ascertained, is to examine for the presence of *hemorrhoids.* Should these be present, search must then be made for any causes, near or remote, which can account for their occurrence, such as, on the one hand, affections of the liver, particularly cirrhosis, or pulmonary and cardiac diseases, with passive congestion of the venous system; or, on the other hand, fæcal impaction as a result of constipation, pregnancy, rectal catarrh, or cancer of the rectum. The presence of the latter affection can generally be satisfactorily ascertained by a careful exploration of the rectum, or of the vagina in women; while, as regards the influence of a disease of the liver or lungs in the production of the hemorrhage, the best guide is the amount of ascites as compared with the development of the dropsy elsewhere.

If no decision can be reached by the methods just indicated, the inquiry must extend to the next most frequent causes of intestinal hemorrhage, such as poisoning, wounds, foreign bodies, and, when these are not indicated by the history of the case, *intestinal ulcers.* The previous history will afford information of the greatest importance in the diagnosis of the latter affection. More or less marked disturbance in the first stages of digestion and hæmatemesis, occurring simultaneously or with the intestinal hemorrhage, point to the existence of a duodenal ulcer, while the presence of indubitable symptoms of tuberculosis or typhoid fever will indicate ulcerations of a corresponding character.

The intestinal hemorrhage which occurs during the course of severe intermittent fevers[1] may readily be mistaken, if the fever be quotidian and the intermissions have become indistinct, for the intestinal hemorrhage of typhoid fever; but the rapid effect of quinine in such cases will render the diagnosis decisive.

When the hemorrhage occurs in a *syphilitic patient*, we should bear in mind the possibility of a specific ulceration, or of an amyloid degeneration of the intestinal mucous membrane; the latter condition is to be inferred with the more probability when the liver and the spleen are enlarged and the urine is albuminous.

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[1] See Frerichs, l. c., S. 358.

We pass now to the rarer causes of intestinal hemorrhage, viz., invagination with the symptoms of ileus, aneurisms, vicarious hemorrhage, etc. In many cases definite information is afforded by the condition of the skin. In the so-called *purpura hemorrhagica*, no other internal organ is so frequently the seat of hemorrhage as the intestine.

I have recently treated a case of this kind, and I give below a brief description of it, not only because it shows how an uncertainty in diagnosis may be cleared up by the cutaneous symptoms, but also because it affords an excellent illustration of the rare complication with *urticaria and affection of the joints*, to which Scheby-Buch has lately called attention.

Meck, a turner, thirty-six years of age, and previously healthy (with the exception of a pulmonary affection several years ago, from which he entirely recovered). Four months ago, without any apparent cause, *red spots made their appearance upon the skin* of the lower extremities, and the limbs became so much swollen that by the third day he was no longer able to stand. *At the same time diarrhœa occurred*—the bowels having previously been quite regular—and after the fourteenth day *tenesmus with passage of blood*, since which time blood, mucus, and matters resembling pus, have been frequently discharged. These symptoms were accompanied by vomiting without any blood, and by pains confined to the lower portion of the abdomen, on the left side. No fever, but at night, occasionally, profuse perspiration. Appetite poor only at the time of the pains; at other times moderate, yet patient is considerably emaciated. No signs of any chest trouble, or of any derangement of the nervous system, except occasional attacks of faintness. The spots alternately appeared and disappeared. There was nothing about his diet or the condition of his dwelling that was at all undesirable; but of late he had been working somewhat harder than usual.

On admission into the clinic, reddish brown petechiæ, with a tinge of yellow, from the size of a linseed to that of a shilling, and some of them confluent, were found in large numbers upon the lower extremities, and a few upon the arms. Some signs of catarrh in the lungs, heart normal, no blood and mucus in the stools, and the urine free from albumen or sugar. Temperature normal. Four days afterwards *swelling of the left hand and some pain in the abdomen* occurred, followed two days later by swelling of the calves and an eruption of new hemorrhagic spots in that part. *The left calf was sensitive to pressure;* shortly afterwards swelling of the arms and palms of the hands, with numerous quite circumscribed soft tumefactions, half the size of a walnut, upon both arms, over which the skin was of a normal appearance. *The stools contained a little reddish-brown mucus.*

Four days after this, *pain in the left knee-joint*, red spots upon both lower limbs, with disappearance of the nodular tumefactions on the arms. Three days later both thighs became covered at various points with *itching, blotchy elevations of the skin, as large as from a pea up to ten times this size, and generally of a white color, but at some places reddened at the top.* The patient recollected that at the beginning of his disease he had had symptoms of a somewhat similar character. *Extension of the urticarial eruption*, within the next few days, *over the upper extremities, trunk, and head; œdema of the eyelids.* The eruption is now almost everywhere of a bright- or dark-red color, and the skin of a spotted appearance. In the upright posture the skin of the lower extremities presented a peculiar appearance, resembling a map of dark-blue alternated with brick-colored markings, the latter being slightly elevated and itching, the former flattened. Three days afterwards the eruption disappeared. Then, on successive days, in the same order, recurred: first, severe pains in the abdomen; then fresh petechiæ; then violent diarrhœa; and afterwards, again, new purpural spots.

Six days afterwards, in the night, *six stools, with considerable tenesmus and blood, and the appearance of fresh petechiæ.* In the morning, again, six bloody stools: then, no more blood, but severe abdominal pains for another day; and then, for three days, strong fever, without any demonstrable local disturbances (104.4; 102.7; 103.4; 99.8; 102.6); then pain occurred in the lower jaw; no lesion of gums. The urine, which has hitherto been free from albumen, now shows the presence of a moderate quantity. Notwithstanding the occurrence of a new purpural eruption, the patient left the hospital of his own accord, *uncured.* The treatment had consisted of Haller's acid elixir[1] in decoction of bark, quinine and iron, tannin, extract of ergot; for the intestinal bleeding, liquor ferri perchloridi; and for the diarrhœa, tannin and opium. *None of these remedies had any effect worth mentioning upon the symptoms, and yet the patient, during his stay in the hospital, had gained eleven and a half pounds in weight.*

We have still, in conclusion, to analyze the diagnosis of another form of intestinal hemorrhage which has been repeatedly observed of late, viz., that produced by embolism of the superior mesenteric artery. Unfortunately, the number of cases described hitherto (about twenty) is so small, that a certain diagnosis of the affection is out of the question. Nevertheless, it is safe to say that, *when conditions are known to exist which can give rise to embolism*, and particularly *when embolism can be proved to have previously taken place in other organs*, a probable diagnosis of embolism of the superior mesenteric artery can be made, if *abdominal pains*, *bloody stools*, and *symptoms of peritonitis*, especially masses of exudation in the abdomen, are present.

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[1] A mixture of sulphuric acid and alcohol, one part to three.—TRANS.

In all the cases observed up to the present time the source of the embolus could be demonstrated without difficulty, generally in the form of an acute or chronic endocarditis; in two cases (Cohn) hemorrhagic nodules were found between the folds of the mesentery, and in one of the cases could even be felt. Bloody stools—the most important diagnostic sign—have, unfortunately, been noticed in only a minority of instances. Our means of diagnosticating the lesion in question are, therefore, as yet very imperfect. In the four cases of embolism of the mesenteric artery examined after death, in Erlangen (see Faber, l. c.), the affection was not diagnosticated, and not even suspected; while a differential diagnosis between embolism of the superior and that of the inferior mesenteric arteries must be regarded as hopeless, notwithstanding the distinction which has been supposed to exist, that in the latter affection *fresh* blood continually oozes from the rectum, and that the pain is more confined to the region of the rectum.

Prognosis.

Whatever its source, intestinal hemorrhage, if at all profuse, is always a critical symptom. It is, of course, true that long-continued, periodically-recurring hemorrhages are often borne remarkably well, and that even a relief to the distress of the patient may be expected when the bleeding is preceded by intense portal congestion, such as occurs in cirrhosis of the liver; in fact, the popular German term for hemorrhoids—flux from the golden vein (Goldaderfluss)—shows how much reliance has been placed upon the discharge of the "morbidly-altered" blood in hemorrhoidal affections. Even under such unfavorable circumstances as typhoid fever, a hemorrhage has been regarded by many physicians as propitious,[1] because its occurrence is often immediately followed, not only by the patient's revival from a lethargic condition, but also by an unquestionable improvement in his general symptoms.

But as soon as the hemorrhage becomes more copious, whatever its source, it must always excite the most intense anxiety on the part of the physician. For, aside from the fact that even apparently favorable hemorrhages may ultimately prove dangerous from the direct loss of nutritive material and the resulting anæmic symptoms, a recurrence of the hemorrhage sooner or later is always to be feared, as a glance at the etiology of the intestinal hemorrhages will show. In most cases, moreover, it is impossible, owing to the uncertainty of the remedies at our disposal, to tell how long the hemorrhage will last.

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[1] See Liebermeister on this subject, Vol. I. of this work, pp. 148, 149.

Hemorrhages from large vessels are more dangerous than capillary bleedings ; but, in estimating the danger in a given case, our best guide, aside from the quantity of blood lost, will be the condition of the patient's strength and the nature of the fundamental disease which has occasioned the hemorrhage, although, of course, the comparative risks in the different affections do not admit of a formal tabulation.

Treatment.

The same principles of treatment which were laid down for gastric hemorrhages will apply also in general to the case of intestinal bleedings, and, therefore, for the sake of avoiding repetitions, we refer to the article on Hæmatemesis, in regard to the special advantages of the various remedies and the indications for their use.

As in gastric, so also in intestinal hemorrhage, the primary indication is *rest*, and this not only for the whole body, but particularly for the bleeding intestine. The latter object will be best attained by *entire abstinence from food*, and by the administration of *opium* when there is evidently an increase of the peristaltic movements of the intestine.

Although experiments have hitherto failed to confirm what has been proved in thousands of cases in which this drug has been used therapeutically, viz., that *opium exerts a quieting influence upon the movements of the intestine*—Nasse, in fact, having observed that, at least immediately after its administration, the drug acts as an intestinal stimulant—and although it is in the highest degree probable that opium at first increases the blood pressure; yet practically, *when one bloody stool rapidly succeeds another*, the use of opium can hardly be dispensed with. Still it should not be forgotten that any harm which may result from this increase of the blood pressure will be more than counter-balanced if we can prevent the rapid removal of the blood from the seat of hemorrhage, and thus, by giving the blood time to coagulate in the intestine, effect a natural closure of the bleeding point.

For the purpose of producing *derivation of the blood* from the seat of hemorrhage, large mustard poultices to the abdomen or similar measures may be employed (see p. 289).

Of the various remedies recommended for a direct stanching of the bleeding, the only one I can recommend is *ergotine*, administered hypodermically in doses of a grain and a half—at least, it is the only one from which I have seen unquestionable results. Moreover, I give *alum whey* as a drink, and for its astringent effects the solution of the chloride of iron, from five to eight drops at a dose in water, or in a decoction of marshmallow ; but as far as any *styptic* action from the latter is concerned, I entertain the same doubts as were expressed in the article on Gastric Hemorrhage. In a similar manner the acetate of lead, tannin, etc., also act as astringents. These astringents promise better results when the hemorrhage proceeds from the large rather than from the small intestine, because, in the former case, by their administration in the form of injections, they can be brought into immediate contact with the bleeding part (one to two per cent. solutions of tannin or chloride of iron).

The styptic most in favor is *ice*, applied externally, either in the form of large frozen compresses (see foot-note, p. 391) or inclosed in large india-rubber bags. However heretical it may sound, I cannot give a very favorable opinion of this remedy. At best, the degree of cold thus produced in the intestinal mucous membrane is trifling, and, even if it were more considerable, we should have to fear what the experiments of Caliburces have shown to occur, when the normal temperature of the body falls to a considerable degree, viz., that very active intestinal movements might be excited. More reliance is to be placed, in my opinion, upon the use of *ice-cold injections* when the hemorrhage proceeds from the large intestine.

Should the weakness of the patient increase, and alarming attacks of *syncope* occur, restoratives may be employed, particularly ether used hypodermically. In reference to this point, and to the dangers of *transfusion*, the reader is referred to the more complete consideration of these subjects, under the head of Gastric Hemorrhage.

In regard to the *after treatment*, it is, above all, important that the intestinal mucous membrane should be spared as much as possible. At all events, entire abstinence from solid food is to be insisted upon for some time, and only the most easily digestible diet, such as milk, meat-solution, etc., should be allowed. It is well, also, to continue for some time the use of astringents. But in all cases the most important indication is to prevent the occurrence of relapses, by measures directed to the fundamental disease. Unfortunately, however, in many cases of intestinal hemorrhage, the prospect of success in this direction is not very encouraging. The special treatment requisite in cases of cirrhosis of the liver, typhoid fever, etc., cannot, of course, be considered here; but this much may be said, that striking results may be obtained by the administration of antidotes in cases of poisoning, and by the use of quinine in the alarming hemorrhages which occur during the course of intermittent fever; moreover, that in anomalous menstruation the bleeding should be restored to the right channel, and that in hemorrhoids and chronic constipation a regulation of the diet and of the evacuations of the bowels are requisite in addition to the merely local treatment of the phlebectases. The question as to how much success is to be expected from the treatment of ulcerations, has already been discussed at length in the chapter on Intestinal Ulcerations (see p. 423). For the intestinal hemorrhages which occur in purpura, the preparations of bark, acids, iron, ergot, etc., have been regarded as indicated by the cause of the hemorrhage. In the so-called "mechanical" hemorrhages, due to an obstruction in the circulation, a relapse may almost always be expected, and even death, when the bleeding is produced by embolism of the superior mesenteric artery. In most of these hemorrhages, occasioned by disturbances of circulation, active stimulation of the heart is required by the causal indication.

Enteralgia. Colica. Enterodynia. Colic.

For a very full account of the older literature s. *Ploucquet.* lit. med. dig. 1805.—*Thom. Willis* op. omn. Tom. II. p. II. Cap. XV. p. 320. ff. De Passione colica. Genf. 1680.—*Sauvages*, Nosol. methodica. II. 99. ff. 1768, distinguishes 22 varieties of colic.—*Tanquerel des Planches*, Traité des maladies de plomb. 1839.—*Romberg*, Nervenkrankheiten. 1857. S. 160–175.—*Henoch*, Klinik der Unterleibskrankheiten. Bd. III. 1858. 1858. S. 1. ff.—*Wertheimber*, Ueber die Kolik im Kindesalter. Deutsches Archiv für klinische Medicin. 1866. I. S. 225.—*Oppolzer*, Klinischer Vortrag. Wiener medic. Wochenschrift. 1867. S. 724, 742, 755, 773, 805, 853, 883, 964, 979, 1044.—*Norris, W.*, Neuralgia in the bowels. Cure by arsenic. Brit. Med. Journal. 1868.—*Eulenburg*, Lehrbuch der functionellen Nervenkrankheiten. 1871. S. 200.—*Kussmaul und Maier*, Zur patholog. Anat. des chronischen Saturnismus. Deutsches Archiv für klinische Medicin. IX. S. 283.—*Eulenburg und Guttmann*, Pathologie des Sympathicus. 1873. S. 124.—*Beard* and *Rockwell*, Electricity. 1874.

Corresponding to the definition given for the neuroses of the stomach in the introduction to the chapter on Gastralgia, we understand by *enteralgia* those morbid conditions *which are directly referable to the sensitive sphere of the intestinal nerves; which manifest themselves in an excessive irritability of the latter; and in which there is anatomically nothing to be found that contraindicates the exclusive limitation of the disease to the nervous system.* We shall exclude from consideration in this chapter, therefore, all those colicky pains which occur in intestinal diseases attended, like cancer and ulcer of the intestine, by pathologico-anatomical changes in the structure of the wall of the bowel.

We know so little at present in regard to the movements of the intestines and in regard to the nerve-tracks along which the motor influence is transmitted, that I shall not venture to decide the question whether the spasmodic contraction of the intestinal wall, so frequently noticed during the attacks of pain, is of the nature of a neurosis or merely a reflex symptom of the colic.

Etiology.

The causation of enteralgia will be more readily understood if we classify the very numerous conditions which give rise to this symptom, under two heads: the first including the colics which are occasioned by *abnormal irritants in the intestinal canal*, and the other those which are due to *an abnormal irritability of the intestinal nerves.* In many cases the colic is the combined result of both these causes being more intense than one would expect, from the nature of the irritant present in the intestinal canal—for example, the severe colic excited in hysterical patients by an accumulation of gas.

To the first category—*the colics depending upon abnormal irritants*—belong the colicky pains excited by *worms*, which are irritated by certain articles of food. Abnormal irritations may be produced also by *foreign bodies*, kernels of fruits, particular kinds of food, such as mushrooms, shell fish, etc., fæcal concretions and gall stones, the latter, when they are of large size, sometimes giving rise to long-continued colic during their passage through the intestine. (See Frerichs, Leberkrankheiten. II. S. 503.)

The same symptom may occur also in *fæcal impaction*, as a result of the intense irritation produced by the distention of the bowel, and by the continued pressure upon the intestinal nerves. Constipation may also act as an indirect cause of colic by the retention of gases which it occasions above the fæcal accumulation. When the gas becomes inclosed between an impacted fæcal mass below, and a descending mass of fæces above, it gradually distends this circumscribed portion of the bowel, and excites severe pains, which, however, disappear as soon as the flatus escapes. This "wind colic" is particularly frequent and distressing in young children, at which age, moreover, colic from other causes is also a very common symptom. In new-born children, as Sauvages has pointed out, colic may be produced by retention of the meconium. Generally, however, in children as well as in adults, the colic, as already remarked, is evidently due to the use of some fermentable article of food (in adults, cabbage or other vegetables, new beer, etc.), which, by its abnormal decomposition, produces gaseous distention of the bowel and traction upon the intestinal nerves.

This is probably the explanation of all those cases in which the attack of colic has been preceded by the use of spoiled or fermenting articles of food. The same symptom may, however, occasionally be produced by otherwise perfectly sound ingesta, when they are taken very cold or in excessive quantities, or if they are coarse and indigestible, as is the case with many vegetables, fruits, etc.

Another very frequent cause of colic is an *exposure to cold*, particularly when the exposure affects the skin of the abdomen or feet. The neuralgia (colica rheumatica) which occurs under these circumstances is probably produced in this way: the suddenly cooled blood upon the surface of the body, entering the intestinal vessels under the strong pressure induced by the collateral hyperæmia, acts as an abnormal irritant to the sensitive nerves (Colin) which run in the sheaths of the intestinal arteries. Many *cathartics*, such as senna, which are well known to excite colicky pains without inducing inflammation, may also be included among the causes of colic.

While, as in the preceding cases, the irritation of the intestinal nerves, by which the enteralgia is produced, usually starts in the intestinal wall itself, in other instances the irritation may proceed from the more centrally situated portions of the nerves. At least this seems to be the explanation of the very rare colics, which occur in diseases of the spinal cord, and of the more frequent radiated enteralgias met with in affections of the liver, kidneys, uterus, ovaries, etc. Upon this point we possess anatomical evidence of quite a decisive character, in the results of some autopsies made upon persons who have died from *lead poisoning*.[1] Of all the remedies used in medicine, there are none which are so notoriously apt to produce colic as *lead*, and, to a less degree, the *preparations of copper*.

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[1] Tanquérel reports one case (No. 25) in which the ganglia of the abdominal sympathetic were enlarged to two or three times their natural size. Ségond, in the endemic colic in Cayenne—undoubtedly an endemic of lead colic—repeatedly found hypertrophy and induration of the ganglia and filaments of the sympathetic (see Romberg, Nervenkrankheiten p. 162). Recently, Kussmaul and Maier, in an individual who had died from chronic saturnism, found proliferation and sclerosis of the connective tissue septa in several of the ganglia of the sympathetic, and proliferative thickening of the connective tissue in the sheaths of the vessels, especially the small arteries of the intestinal submucosa.

The action of the former has been carefully studied. Tanquérel des Planches, in his epoch-making work on lead diseases, expressed the opinion that lead colic was due exclusively to an affection of the sympathetic; and although such lesions have been found in only a few instances, his theory must be accepted at the present day as a very plausible one, especially in view of the fact that Heubel, who induced lead colic in animals experimentally, has proved that the nerve tissue has a greater affinity for lead than is shown by any other organ which can be concerned in the production of the symptoms of lead poisoning. Lead may enter the body in a great variety of ways, but, as is well known, the most frequent sufferers from saturnism and lead colic are those persons who use preparations of lead in their business—for example, painters, potters, and especially the workmen in white lead and red lead factories. As this subject is fully considered in another part of this work (Toxicology, last volume of this cyclopædia), it is unnecessary to go into any further details here.

The forms of colic last mentioned introduce us to our second subdivision of enteralgia, viz., that in which we have to deal with a *change in the normal condition—a hyperæsthesia—of the intestinal nerves*, and in which, therefore, the element of abnormal irritation plays but a subordinate part. At the head of the causes included under this category stands *hysteria.* This neurosis, which is characterized by a perverse irritability of the entire nervous system, gives rise not only, as we have previously seen, to neuralgia of the stomach, but also to enteralgia. The colics which occur in rheumatic patients are probably also to be referred to an abnormal irritability produced by the impaired nutrition of the intestinal nerves.

As in gastralgia, so also in enteralgia, the cause frequently eludes detection.

Symptomatology.

The attack of colic may either be preceded by premonitory symptoms, such as nausea, tympanites, rumbling and slight griping pains, or it may develop at once in its full intensity without any warning. The pains are of a tearing, cutting, and most frequently of a *griping* character, and, although they may shoot in various directions, they are usually concentrated in the region of the umbilicus. The patient is generally restless; anxiety and an expression of pain are depicted upon his features ; the extremities and face are cool and covered with a cold sweat ; the pulse is usually small, hard, and, at least in lead colic, slow. The patient either lies quiet with the body bent together, or he rolls about in pain, and seeks to relieve this distress by pressing his hands or some solid object against the abdomen, or by lying upon his stomach. Frequently, however, the abdomen is painful on external pressure, and may even be hyperæsthetic. The appearance of the abdomen differs very much in different cases ; the integuments are tense, sometimes retracted, at other times distended, either over the whole surface or occasionally only at particular parts. In the latter case the local distention or retraction may be occasioned by the imprisonment of gas in certain portions of the intestine or by spasmodic contraction at particular points. The participation of the abdominal muscles in the spasm is clearly shown by their hardness and tension, and in some instances by the retraction of the testicle when the cremaster muscle also becomes contracted.

That the pain is a true neuralgia is apparent, not only from what has already been said, but also from its periodic character —although this feature of the pain is not so uniformly present as in other neuralgias—and from the sudden cessation of the pains, followed by a feeling of complete relief. Not unfrequently the attack terminates with a movement of the bowels or an escape of flatus in an upward or downward direction.

Among the *concomitant symptoms* may be mentioned : vomiting, cardialgia, oppression of breathing, hiccough, tenesmus, urgent micturition, priapism, giddiness, fainting, tremors or actual convulsions. Constipation is generally present in cases of non-inflammatory colic, and, when the attack is due to lead poisoning, this symptom may last for days or weeks.

In young children the pain of colic is manifested by cries, restlessness, and retraction of the limbs. The child refuses to nurse, or lets go of the nipple as soon as it takes hold of it.

The duration of colic is extremely variable ; it may last only a few moments or for days, with intervening remissions, and is very liable to return.

With our present views enteralgia must be regarded as a neurosis of the sympathetic nerve. At least the complex of symptoms just mentioned, as well as the post-mortem examinations above referred to, seems to point in this direction, and recently attempts have been made to throw additional light upon the subject by bringing the symptoms into connection with certain physiological facts.[1] The constipation, for instance, may be explained by the inhibitory action of the splanchnic nerve upon the peristaltic movements of the intestine (Pflüger), and probably also the scanty urine in lead colic by the controlling influence of the same nerve through its renal branch upon the renal secretion (Eckhard, Knoll). The element of pain finds its physiological analogue in the pain which occurs on division of the splanchnic nerves (Ludwig and Haffter),[2] and may also be explained by the fact that the arteries of the abdominal viscera are specially provided with sensitive nerves which form a network around the vessels (Colin).[3] Finally, the retardation of the pulse may be naturally explained by the reflex inhibition of the cardiac movements, which occurs when the vagus is irritated, as is illustrated by the well-known experiments of Goltz, in which the same symptom was produced by blows upon the epigastrium. Inasmuch, also, as irritation of the vagus is known to produce violent movements of the intestine (Sanders Ezn.),[4] it is probable that the spasm of the bowel in colic is also to be referred to similar reflex processes.

Diagnosis.

The diagnosis of colic, when the chief features of the disease are well marked, usually presents no difficulties. On the other hand, it is by no means easy to distinguish between a nervous colic and the pains which proceed from inflammatory or ulcerative processes in the intestine. The distinction generally relied upon, viz., that pressure upon the abdomen increases the pain of inflammation and diminishes that of pure colic, is unfortunately a deceptive one, because in intestinal ulceration the patient sometimes experiences relief from pressure, while in lead colic the pains are frequently aggravated by the same means. The general rule, however, still holds good that, when the pain is increased by deep pressure, inflammation rather than enteralgia is indicated, while if the attacks of pain occur suddenly in the midst of good health, and the severe pain be followed, without treatment, by entire relief, enteralgia is indicated. Attention, however, should be directed to the coexisting symptoms of other diseases, and particularly to whether the attack has been preceded by any injurious influences which may have occasioned the colic.

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[1] See *Heubel*, Pathogenese und Symptome der chron. Bleivergiftung. 1871. *Eulenburg* and *Guttmann*, Die Pathologie des Sympathicus. 1873.

[2] *Henle* and *Pfeuffer*, Zeitschr. für rationelle medicin. Neue Folge. 4. S. 322.

[3] Comptes Rendus. 65. p. 403.

[4] Centralbl. für die medic. Wissenchaften. 1871. S. 479.

As regards the *special* causes of the affection, if the attack begin with uncomfortable sensations in the epigastrium and with vomiting, the chances are that the colic has been produced by *indigestible food*, and the vomited matters should always be inspected, in the hope of finding portions of the offending ingesta. If the colic depend upon an *accumulation of fæces*, the diagnosis may be made from the antecedent constipation or sluggishness of the bowels, the detection of nodular masses in the abdomen, the presence of conditions which are favorable to the occurrence of constipation (*e. g.*, pregnancy), and finally from the relief afforded by a movement of the bowels. Colic, arising from an excessive accumulation of *intestinal gases*, may be diagnosticated by the considerable distention of the abdomen—sufficient in some cases to interfere with the respiration—by the brisk rumbling along the intestine, and the momentary improvement of the symptoms when the flatus escapes. When the colic is excited by the presence of *worms* in the intestinal canal, we are to be guided by the previous history of uncomfortable sensations in the abdomen, the craving for food, and the existence of a great variety of nervous derangements. The diagnosis in such cases will, of course, be clearer if round worms or fragments of taniæ have escaped in the stools.

When the colic is due to *rheumatic* influences, the symptoms are rarely of a distinctive character; our only reliance in such cases must be the positive absence of other causes, and the fact that the outbreak of the enteralgia was immediately preceded by a marked *exposure to cold.*

The colic which occurs in *hysterical patients* is sometimes difficult to distinguish from abdominal inflammation, on account of the cutaneous hyperæsthesia in these cases, which renders pressure very painful, and may thus counterfeit peritonitis, as is shown by the following striking case reported by Henoch.

A young hysterical woman was attacked with extremely severe abdominal pains, which increased from hour to hour, and radiated from the umbilical region in all directions. The pains were continuous, but at times increased to an extraordinary violence. Any pressure upon the soft undistended abdomen was painful, and rough handling of the skin of the abdomen excited extreme distress. Painful and urgent micturition, pulse unusually rapid, features sunken, extremities cool. The attack lasted in its full intensity for forty-eight hours, then gradually declined, and finally ceased entirely on the fourth day, leaving the patient in a state of extreme prostration. After a few months the same scene recurred.

Under such circumstances the presence of the globus hystericus, spasms, and other symptoms of hysteria, the observation that deep pressure is not materially more painful than the most superficial pressure—a symptom to which Watson has called very particular attention—the age of the patient, etc., will protect against mistakes. Still, in view of the similarity between the symptoms of colic and those of a commencing peritonitis, we should never forget to search for any causes which might occasion the latter, and particularly to carefully examine the abdominal rings for an incarcerated hernia.

Lead colic is usually distinctly charaterized by obstinate constipation, with a tense, or, it may be, retracted abdominal wall, scanty urine, hardness, and slowness of the pulse, a bluish-gray line upon the gums, paralysis, etc. ; still in lead colic also the *antecedent history* is of far more importance than the symptoms of the attack.

The particular portion of the intestine which is chiefly affected can very rarely be determined. The situation of the pain in the umbilical region is certainly no indication that the small intestine is the part affected, because Bamberger has found the umbilical region painful in the great majority of cases of dysentery as well as in very many other intestinal affections attended by colic.

When, however, the pain is clearly limited to the hypogastrium and sacral region, and is accompanied by a "bearing-down sensation" in the rectum and its vicinity, it is safe to infer that the sympathetic nerve filaments of the hemorrhoidal plexus, which are distributed to the inferior portion of the rectum, are affected—a form of neuralgia of the sympathetic which Romberg[1] was the first to describe, and to which he gave the name "hyperæsthesia plexus hypogastrici." Aside from its occurrence in the not infrequent menstrual colic of women, it is met with most commonly in connection with hemorrhoidal disease of the rectum, and then may receive the special designation of "hemorrhoidal colic."

Prognosis.

If we leave out of account the colics, which are produced by local disease of the intestine, and in which danger may arise from the fundamental disease, such as ulceration, strangulated hernia, etc., the prognosis of the colic itself may be regarded as *favorable.* In very rare cases, however, death may occur at the height of the attack from rupture of the intestine, which has been excessively distended by gas (Oppolzer), or from convulsions, as happened in a case reported by Wertheimber, in which round worms, knotted together in a mass as large as a fist, were found in the small intestine. In judging of the course which the affection is likely to pursue, it should also be borne in mind that the colic in many cases is very apt to recur.

Treatment.

The removal of the *causes* which underlie the occurrence of the colic constitutes of course the most important indication, and all the more because relapses are always to be apprehended. This indication can be best carried out in most cases by the administration of *cathartics*, not only when the colic has been produced by fæcal accumulation, or by the use of irritating, fermenting, flatulent articles of food, but also even in cases of hysterical enteralgia, because here also the violent reaction of the intestinal nerves may be immediately excited by the presence of abnormal ingesta. In lead colic, moreover, purgatives form an important part of the highly praised Traitement de la Charité and of the various modifications of this treatment. Usually, however, at least in the severer forms of enteralgia, it is best to first treat the attack itself, and then to carry out the above-mentioned causal indication immediately after the attack has subsided. In all cases, with the exception of lead colic, *mild* cathartics are to be preferred, because the more powerful ones are liable to directly aggravate the colic. This precaution was observed as long ago as the time of Sauvages, who recommended only the long-continued use of the oil of sweet almonds in large doses. In place of this, we may use also castor-oil, rhubarb, or calomel, or we may evacuate the bowels by means of enemata.

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[1] l.c. S. 174.

When the colic is produced by an accumulation of gas, relief may be sometimes afforded by expelling the gas from the bowel, either upwards or downwards, by means of cautious pressure upon the abdomen. In such cases, moreover, *carminatives* have been found useful by promoting the peristaltic movements ; perhaps also, remedies, which check fermentation in the intestine, may be used with advantage : capsicum, chamomile, caraway-seeds, peppermint leaves, from half a teaspoonful to a teaspoonful to a cup of hot water. These infusions are useful, also, in *colic from exposure to cold,* because they seem to aid the desirable diaphoretic action, which can be induced by covering the patient with blankets. Heated stones, poultices, etc., are also serviceable—in fact, the majority of attacks of colic are benefited by the application of heat. In *hysterical* cases the derivative treatment may be supplemented by enemata of asafœtida (a drachm and a half) or valerian (two drachms and a half to the injection), and in some cases these remedies unquestionably act very favorably. When the colic is excited by the presence of worms, anthelmintics should of course be given in addition to cathartics, and in *hemorrhoidal* colic leeches to the anus may be required by the indicatio causalis.

As in all neuralgias, our main, and in fact only, reliance for the immediate relief of pain, is *narcotics*—generally opium.

The usual treatment is to first alleviate the pain by laudanum or a hypodermic injection of morphine, then to give a cathartic, and afterwards more opium if necessary. The usually constipating action of opium need not deter us from using the drug where an evacuation of the bowels is desirable, because we may reasonably hope by its use to relax the spasmodic contraction which is very commonly present in some portions of the intestines and which acts as an obstacle to the propulsion of the ingesta. Experience has shown—at all events, particularly in cases of lead colic—that it is the administration of opium which first produces a movement of the bowels and a relaxation of the abdomen.

Still more rational is the use of atropine, which, as shown by von Bezold and Bloebaum, diminishes or even abolishes the irritability of the intestinal nerves, and thus places the intestine in a condition of rest. This remedy deserves a more extended trial in colic, in view of the excellent results obtained by Wertheimber, in the colic of children, with the extract of belladonna dissolved in bitter almond water (one grain to the ounce, eight drops every one or two hours), while, by the continued use of the drops (six drops two or three times a day), he always succeeded in overcoming even obstinate constipation.

Whether *electricity* is as serviceable in cases of colic as in cardialgia, it is impossible to say. It may be employed with some benefit for one of the symptoms connected with the colic—the *meteorism*—as I can confirm from my own experience When the meteorism is so highly developed as to threaten rupture of the intestine, it may be necessary to puncture the bowel with an exploring needle, as has recently been frequently done with favorable results.[1]

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[1] See the discussion in the Paris Academy of Medicine, *Canstatt's* Jahresber. 1871, p. 151—especially the statement of Fonssagrives, that he has punctured eighty times without bad results.

Solutions of Continuity.

Rupture of the Intestine. Enterorrhexis.

Old literature. s. *Ploucquet's* Lit. med. digesta under Intestini Ruptura spec.: *Clauderus*, Cervus venatorem modo subito et raro occidens. Ruptures in the Ileum, Colon and Duodenum. Eph. Nat. Cur. Dec. II. A. VII. Obs. 182.—*Wepfer*, Zerreissung des Jejunums und Ileums. Eph. Nat. Cur. Dec. II. A. X. Obs. 170, 171, u. 172.—*Bonet*, Sepulchr. Lib. III. Sect. XIV. Add. Obs. 3.—*Ebermaier*, Freiwillige Durchlöcherung des Magens und Darmkanals. Fall I. Casper's Wochenschr. 1835. S. 167.—*Davies*, On the diagnosis of rupture of the stomach and intestines. Lond. Gaz. Septbr. 1845.—*Kiwisch*, Spontane Berstung des dünnen Darms, etc. Prager Vierteljahrschrift. 1844. III. S. 28–35.—*Banner*, Rupture of the intestines from external violence. Reports of the Liverpool Pathol. Soc. 1844–45. Ref. Schmidt's Jahrb. S. 234—*Dittrich*, Ueber spontane Zerreissung des Darmkanals. Prager Vierteljahrschrift. 1846. III. S. 121.—*Nathan Ward*, Remarks on strangulated hernia, etc. Lancet. 1856. Ref. Schmidt's Jahrb. XCI. S. 93. ff.—*W. Koster* aus *Polano's* Klinik. Ruptur des Coecums. Nederl. Tijhschr. III. 1859. Schmidt's Jahrb. CV. S. 82.—*Poland*, Contusions of the abdomen, etc. Guy's Hosp. Rep. Ser. 3. Vol. IV. p. 123. et seq. with a compilation of cases; an exhaustive article.—*Schreiber*, Wiener med. Halle. IV. 1863. Ref. Schmidt's Jahrb. Bd. 123. S. 200.—*Wachsmann*, ibid.—*Gutherz*, Berstung der Flexura sigmoidea. Bayer. ärztl. Intelligenzbl. 1863. 48.—*Casper*, Gerichtl. Medicin. 1864. II. § 35. ff.—*A. Poland*, Injury to the abdomen, jejunum torn, etc. Med. Times and Gazette. 1868. Vol. II. S. 445.—*Holland*, Rupture of the jejunum. British Med, Journ. 1873.

As in the chapter on Rupture of the Stomach, so here we shall consider only those solutions of continuity which have not been preceded by destructive processes in the wall of the bowel. The perforations which occur as a complication during the course of ulcerations, poisoning, abscess of the liver, etc., properly belong to the articles on these respective diseases.

True rupture of the intestine is always occasioned by influences of a mechanical nature, which, acting upon the intestinal wall, either *from within or without*, produce a laceration of its tissue. This effect may result from a severe blow, a fall upon the abdomen, a rough attempt at taxis,[1] from being run over, etc.; but in all these cases it is necessary that the violence should be very considerable, while injuries of this character are much more apt to lacerate other abdominal organs than the elastic intestine. Rupture occurs more frequently in the small than in the large intestine. Poland has found, moreover, that in lacerations of the jejunum the rupture generally takes place in the upper portion, near its junction with the horizontal inferior part of the duodenum. The explanation in such cases is simply this, that the latter division of the intestine is firmly connected with the surrounding parts, and therefore, when the upper end of the jejunum is acted upon by the external force and is suddenly dragged from its position, it is unable to yield to the traction, and thus becomes lacerated in the neighborhood of fixed point. In the large intestine, notwithstanding its superficial position, laceration from violence is, upon the whole, rare, because this portion of the bowel is very distensible, and has a firm support in its muscular bands—the tæniæ coli. When rupture from contusion, etc., does occur in the large intestine, we may explain it (Koster) by the fact that the gas, which, in the case of compression of the small intestine, can usually[1] escape in both directions, here finds its exit impeded or entirely prevented by the presence of hard fæcal masses or by the ileo-cæcal valve.

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[1] See Davies, l. c.

The mention of this last mode of rupture naturally brings us to the consideration of those cases in which, independently of any external violence, *laceration of the intestine is produced by an excessive accumulation of gas*[2] *within the bowel.* Under ordinary circumstances such a distention is never so considerable as to produce laceration, because it excites an energetic peristalsis, which shifts the position of the gas and the fæcal masses forward. If, however, the contraction of the bowel be prevented by a paresis of the intestinal muscles, or be counteracted by a stenosis which opposes an insuperable obstacle to the efforts of the muscles, the final result must inevitably be a rupture of the wall of the intestine. The pathological conditions which may induce stenosis are: contracting cicatrices remaining after ulcerations, cancer, incarcerations, invaginations, twistings of the bowel, adjacent tumors, or foreign bodies in the intestine. In the presence of such predisposing causes, it needs only the occurrence of violent contractions above the stenosed part to bring about the final rupture.

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[1] If two convolutions of intestine, lying parallel to each other, are subjected to a considerable degree of compression at both ends, it is conceivable that the excessive distention from intestinal gas may produce a rupture at the point of flexion.

[2] An excessive accumulation of solid *fæcal masses* can hardly ever produce laceration of the bowels, because the irritation excited by these masses will force them forwards, or if for any reason this result is prevented, the impaction will merely produce a further accumulation in the upper portions of the intestine. At all events, the distention takes place so slowly that, in place of rupture, we may expect rather a slow process of gangrene, finally resulting in perforation.

At the *autopsy* the intestine is generally found to be torn transversely. Almost without exception, the laceration is situated opposite the insertion of the mesentery (Dittrich), and in some cases extends clear across the bowel, making a gap several centimetres in width. The less distensible serosa seems to give way first; at least this is what happens when compression is made upon the blown-up intestine in the cadaver (Bochdalek; see Kiwisch, l. c.). When the laceration during life has been incomplete, the serosa and muscularis are found torn, while the mucous membrane remains intact.

Thus in Kiwisch's case (l. c.), besides the rent from puncture, there was also another laceration running all the way across the ileum, penetrating for the most part only as far down as the muscularis, only at points injuring the cellular layer, and covered with ecchymoses. In a case reported by Dittrich (l. c.), an oval transverse laceration was found in the large intestine penetrating all the layers, and, some distance above, another transverse rent, an inch and a half in length, involving merely the peritoneum and the longitudinal muscular bundles, while the very thin mucous membrane, which bulged into the rent, looked as if it were on the point of rupture.

If the laceration extends through the mucosa, the gap may be irregular in shape, and, in consequence of the valvular bulging of the mucosa, seem smaller than the actual transverse rent through the serosa.

The rupture presents, upon the whole, much the same appearance, whether it occurs in the small or large intestine; but in the latter situation there are certain peculiarities, occasioned by the special structure of the part, which require separate mention. While in suddenly occurring ruptures the laceration involves the sacculi and bands equally, this does not seem to be the case when the laceration is produced gradually. When, in consequence of a stenosis of the large intestine, the parts situated above the obstruction have become excessively distended, and their muscular coat hypertrophied, the thickened muscular bands yield to the pressure less readily than the sacculi, which thus become forced between the bands. This bulging and tension on the part of the sacculi may be so great that, as Dittrich, who was the first to call attention to this mechanism, has expressed it, "the mucous membrane, driven by the distending force into these projections, is, as it were, cut through by the edges of the longitudinal muscular bands, forming lacerations varying in length and breadth, on the inner surface of the bowel along the course of the bands." The sacculi, which are thus predisposed to rupture, will therefore be lacerated more readily than the bands, when the tension is increased.

As soon as the fæces escape into the peritoneal cavity, a more or less intense *peritonitis* usually occurs ; in rare instances an unmistakable attempt at healing has been noticed, the opening being partially closed by the projecting edge of the mucosa, or entirely filled by a piece of omentum.

The *symptoms* in cases of rupture of the intestine are those of perforation-peritonitis in general ; pain in the abdomen, collapse, and vomiting usually ensue immediately after the occurrence of laceration, although in certain cases (as in one recently reported by Poland) the collapse and vomiting do not follow immediately, and the patient may be able to get up and walk about. Later the marked signs of peritonitis are found : distention of the abdomen, displacement of the liver dulness from the pressure of the gas which has escaped into the peritoneal cavity, etc. Death may ensue within a few hours, usually during the course of the first or second day, and in rare cases not until after several days or even weeks. As already pointed out, the possibility of a cure should be borne in mind.

A few instances of partial or complete recovery have been reported. Thus, in a case of traumatic rupture of the ileum without wound of the abdominal wall, and in which death occurred sixteen days after the accident, with symptoms of pyæmia, a reparative peritonitis was found, with a fæcal abscess. Poland reports the case of a boy who had been run over and had lain for days in an almost moribund condition, with a tensely swollen abdomen, but who finally recovered after four weeks under the use of opium and ice applications, by which the peritonitis had been limited.

The *prognosis* in cases of supposed rupture of the intestine is manifestly *absolutely bad*, because, even when the gap in the intestine is *completely* occluded by a firm peritoneal exudation, and perfect rest of the intestines has been maintained, death may occur from collapse, as recently happened in a striking case of my own.

The *diagnosis* of rupture of an air-containing abdominal viscus is usually not difficult; on the other hand, it is difficult, and in fact generally impossible, to determine what portion of the digestive tract has been lacerated. At most we can only succeed in distinguishing perforation of the stomach from that of the intestines by means of the history of the case, the bloody character of the vomiting, and the situation of the intense pain; still even here the diagnosis will scarcely rise above the level of a mere conjecture. A determination of the particular portion of the intestine in which the supposed rupture has occurred is usually entirely impracticable, although Poland was so fortunate as to find his bold diagnosis of a rupture of the jejunum confirmed by the autopsy.

The *treatment* of this hopeless condition consists in the prescription of opium, ice, absolute rest, entire abstinence from food, the use of stimulants in collapse, and similar measures indicated by the gravity of the case.

TRANSLATOR'S NOTE. The lecture referred to on page 223 does not contain the recipe for making the meat solution there recommended, but simply states: this solution is prepared by digesting meat with a strongly acid solution of pepsin in hermetically sealed vessels, at a temperature much higher than that of the human stomach. Under this treatment the meat is ultimately reduced to a very fine emulsion, and the solution contains a greater or less quantity of peptones.

CONSTRICTIONS, OCCLUSIONS,

AND

DISPLACEMENTS OF THE INTESTINES.

LEICHTENSTERN.

CONSTRICTIONS, OCCLUSIONS, AND DISPLACEMENTS

OF

THE INTESTINES.

Those treatises which were not accessible to me in the original, but which, for the sake of completeness, must be mentioned here, are marked by an arterisk *.

A Paraeus, Op. chir. Edit. latin. Francof. 1594.—*Schenk*, Obs. med. rar. Francof. 1602. Lib. III.—*F. Plater*, Prax. med. Basil. 1602.—*Fabr. Hildanus*, Opera Francof. 1646.—*D. Sennert*, Prax. med. Lib. III. de ventris morbis. Wittenberg. 1670.—*Sydenham*, Op. univers. Lugd. Bat. ed. 1726. p. 75.—*Th. Bonnet*, Sepulchr. s. anat. pract. Lugd. 1700. Lib. III. p. 29. Lib. II. pp. 13, 14.—*Manget u. Leclerc*, Biblioth. anat. Genev. 1685. T. I. de infimo ventre.—*J. C. Peyer*, Parerg. anat. med. sept. Lugd. Bat. ed. 1736. p. 61.—*Ruysch*, Op. Omn. anat. med. chir. Amstelod. 1721.—Numerous historically important communications in the Ephemerid. medico-phys. Germanic. acad. Caesareo-Leopoldin. naturae. curios. Incipit. ab. anno 1670; also in the Act. erudit. Lipsiae, in the Histoires and Mémoires of the Acad. Roy. des Sciences. Incip. ab anno. 1666.—*Fr. Hoffmann*, Med. rat. syst. Halae. Magdeburg. 1734. T. III. p. 1. C. 4, 16, 17, pp. 2, 11. T. IV. Pars. I. p. 2.—*A. Haller*, Disp. select. ad morb. hist. et cur. Lausannae. 1757. T. III. (Vater, Kuhn, Leidenfrost, Kupffer, Troschel . . .) Disp. chir. T. III. (Schacher, Lavater, Kirschbaum). Disput. anat. T. I. (Walther). T. VII. (Velse).—*de Haën*, Rat. med. Vindob. 1757. Pars. II. C. V, P. VIII. C. V, P. IX. C. V, P. XI. C. III. Rat. med. *continuat.* Tom. II. Pars. II. C. 2.—*van Swieten*, Comment. in H. Boerhaav. aphorism. T. III. p. 106. ff.—*Gallezky*, Abhdlg. v. d. Miserere. Mitau u. Riga. 1767—*Fr. Boissier de Sauvages*, Nosol. meth. Lips. 1796. T. IV.—*Meyer*, Dissert. de strangulat. Argentorati. 1776, u. in den Neuen Sammlungen d. auserlesensten Abhdlgn. etc. f. Wundärzte. XVI. S. 143. ff.—*Morgagni*, De sed. et caus. morb. Ep. 34, 35. Ep. 39. Art. 29. Ep. 54. Art. 11-13.—*D. Rahn*, De pass. iliac. pathol. Halae. 1791.—*J. Lieutaud*, Hist. anat. med. Par. 1767.—*Baillie*, The morb. hum. anat. German by Sömmering. Berl. 1794.—*Ed. Sandifort*, Obs. anat. path. Lugd. Bat. 1777.—*A. Monro*, The morb. anat. of the hum. gullet. Edinb. 1811.—*J. Fr. Meckel*, Hbd. d. path. Anat. Leipz. 1812. Bd. II. Abth. 1.—*A. G. Richter*, Spec. Ther. Berl. 1813. Bd. 4. S. 203.—*Jos. Frank*, Prax. med. Thl. 3. Vol. 2. Sect. 1, mit Angabe einer grossen Zahl von Dissertat. bis zum Jahre

1835.—*Fleischmann*, Leichenöffnungen. Erl. 1815.—*Seckendorff*, Collect. quaed. de strangulat. Lips. 1825.—*Forbes*, Edinb. Med. Comm. Vol. IX. p. 266.—*Monfalcon*, Dict. des sc. méd. T. XXIII. Art. Ileus.—*Vetter*, Encycl. Wörterbuch. Art. Ileus.—*Naumann*, Hdb. Bd. 4. Abth. 1. S. 755.—**Masson*, Th. de Par. 1826.—*A. Cooper*, The anat. etc. of abdom. hernia, by A. Key. Lond. 1827.—*Abercrombie*, Diseases of the stomach, etc., translated into German by Gerh. v. d. Busch. Brem. 1830.—*Cruveilhier*, Traité d'anat. path. gén. T. I. II. u. Anat. path. Atlas u. Text. Livr. VII. Pl. 5, L. XV. Pl. 6, L. XXI. Pl. 6, L. XXII. Pl. 4. 6, L. XXV. Pl 3, L. XXVI. Pl. VI, L. XXXVIII. Pl. 6.—*Albers*, Atl. d. path. Anat. IV. Abth. Bonn. 1832.—*Lobstein*, Lehrb. d. path. Anat. Deutsch v. Neurohr. Stuttg. 1834.—**Bonnet*, Th. d. Par. 1830.—*C. A. Clarus*, De oment. lac. etc. Lips. 1830.—**Testu*, Th. d. Par. 1830.—*Renaud*, Th. d. Par. 1833.—*Philipps*, On intest. obstruct. Med. Chir. Trans. Vol. XXXI. 1.—*Froriep*, Chir. Kupfertafeln. Weimar. 1820–47. bes. Taf. 378.—*Wagner*, Med. Jahrb. d. österr. St. IV. 1833.—*Rokitansky*, ibid. X. 1836. Lehrb. d. path. Anat. 1861. 3. Aufl. 3. Bd. S. 181. ff.—*Durand*, Th. d. Par. 1835.—**Largeau*, Th. d. Par. 1840.—*Höhne*, Diss. de strang. Vratisl. 1841.—*Neussel*, Diss. de strang. Marb. 1841.—*Forcke*, Unters. üb. Il. etc. Leipz. 1843.—*G. Pfeiffer*, De laparot. in volvulo necess. Marb. 1843.—*Canstatt*, Spec. Path. u. Ther. Erl. 1843. 4. Bd. S. 563.—*Chomel*, Gaz. des hôp. 1846.—*Wunderlich*, Hdb. d. Path. u. Therap. 1846. Bd. III.—*Monneret et Fleury*, Comp. V.—*Leubuscher*, Hdb. d. med. Klinik. Lpz. 1859.—*Bamberger*, Virch. Hdb. d. spec. Path. u. Therap. Bd. VI. Abth. 1. S. 398. ff. 1. Aufl. 1855.—*Oppolzer*, Wien. med. Wochenschr. 1852. Deutsche Klinik. 1859.—*Emmert*, D. Unterl.-Brüche, etc. Stuttg. 1857.—*Pirogoff*, Abhdlg. a. d. Geb. d. Heilk. v. e. Gesellsch. prakt. Aerzte in Petersb. 8. Samml. 1852. S. 145.—*Rieux*, Th. d. Par. 1853.—*Virchow*, in s. Arch. V. 1853. S. 334. ff.—*Savopoulo*, Th. d. Par. 1854.—*J. Hutchinson*, Med. Tim. and Gaz. April. 1858.—*Streubel*, Prag. Viertelj.-Schr. 1858. 1. 57.—*Duchaussoy*, Mém. de l'Acad. de Méd. XXIV. 1860.—*Pélous*, Th. de Strassbourg. 1859.—*Copland*, Diction. of pract. med. Lond. 1858. Vol. II.—*Houël*, Mém. cour. par l'acad. de Méd. d. Paris (Noblet). 1860.—*Besnier*, De l'étranglement int. Par. (Coccoz). 1860.—*A. Wachsmuth*, Virch. Arch. 1862. Bd. XXIII. S. 118.—*Küttner*, ibid. XLIII. S. 509. ff.—**Mony*, Th. d. Par. 1860.—*Trousseau*, Clin. méd. 1862. T. II. p. 504.—*H. Lebert*, Traité d'anat. path. gén. et spéc. Atlas u. Text. Tom. II. Pl. 118, 119, 121, 123.—*Henoch*, Klinik d. Unterl.-Krankh. Berlin. 1863.—*S. O. Habershon*, On diseas. of the abdom. Lond. 1862.—*Mosler*, Arch. d. Heilk. 1864.—*Brinton*, On intest. obstruct. Ed. by Thom. Buzzard. Lond. 1867.—*C. H. Fagge*, On intest. obstruct. Guy's Hosp. Rep. XIV.—*Patoir*, Th. d. Par. 1869.—*Servier*, Mém. cour. p. la soc. medico-chir. de Liége. 1870.—*Bryant*, Clinic. lec. on intest. obstr. Times. 1872.—*Wagstaffe*, St. Thom. Hosp. Rep. 1873. p. 179.—*A. v. Hüttenbrenner*, Die Darm-Stenosen im Kindesalter. Habilit.-Schrift. Leipzig. 1875.

LITERATURE OF OCCLUSION AND CONSTRICTION OF THE INTESTINES, WITH REFERENCE TO LAPAROTOMY, ENTEROTOMY, COLOTOMY: *Hévin*, Mém. de l'acad. de chir. Vol.

III. p. 568. Vol. IV. p. 201.—* *Maunoury*, Thèse de Paris. 1819.—*Switzer*, Adnot. ad. Colot. Hafn. 1826.—*Maisonneuve*, Arch. gén. de méd. 4. Sér. Tom. VII. 1845. —*Thüngel*, Langenbeck's Arch. I.—*Cæsar Hawkins*, Medico-chir. Transact. Lond. 1852. XXXV. p. 85—* *Vassor*, Th. de Par. 1852.—*Trousseau*, Gaz. des hôp. 1857. 59. 1862. 69.—*Nélaton-Veillard*, L'Union. 1857. 89, 91, 93.—*Chassaignac*, Gaz. des hôp. 1855, 1859.—*Heidinger*, Diss. Inaug. Dorpat. 1861.—*Hacken*, Diss. Inaug. 1861. Dorpat.—*Adelmann*, Prag. Vierteljahrschrift. 1863. 2. S. 29. ff.—*Fräntzel*, Virch. Arch. XLIX. 164.—*Delaporte*, Th. d. Par. 1872.—*Whitall*, New York Med. Journ. 1873.—*P. Teale*, Lancet. 1857. Vol. I. p. 369.—*Erskine Mason*, Amer. Journ. of Med. Sc. Oct. 1873.—*John Ashurst*, Americ. Journ. 1874. Extr. dans les Arch. gén. de méd. Jan. 1875.

In addition we refer to the bibliography given in connection with the different kinds of occlusion and constriction, and also to the citations in the text.

Introduction. History.

The numerous and varied processes which give rise to occlusion of the lumen of the intestine show themselves clinically by a train of symptoms, which, in the main, does not vary, was recognized in the earliest ages, and known under different names, such as ileus or passio iliaca, volvulus, chordapsus, miserere, Darmwinde (gripes), Darmelend (colic), etc. Like most of the titles of symptomatic medicine, the term ileus was applied during many centuries to diseases that were anatomically very different, and complete unanimity in the use and definition of the word was not arrived at. This is sufficiently shown by the following sketch of the history of ileus.

The word ileus or volvulus was used by the medical writers of antiquity as a collective term for different diseases of the abdomen. Under this title we sometimes find a complete description of the symptoms of occlusion of the intestine[1], sometimes simple colics, vomiting, and purging, peritonitis, typhlitis, etc. From the writings of Hippocrates[2] it appears that even distention of the abdomen, as by ascites in diseases of the liver (ileus auriginosus), or by tympanites in typhoid fevers (ileus sanguineus), was termed ileus, and it is therefore not to be wondered at that we sometimes find this described, in the writings of the ancients, among the chronic diseases, or as a morbus creberrimus. Those are certainly in error who infer, from the etymology of the word,[1] that the ancients had an idea of occlusion of the intestine by "contortion of the bowels." The term ileus means only that the disease occupies the "convoluted intestine," and is also appropriate on account of the accompanying, peculiar, twisting pains ("dolor convolvens se") under which the patient writhes.[2]

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[1] *Hippokrates*, De morb. Lib. III. Ed. Kühn, T. II. p. 304.—*Aretæus*, Ed. Kühn, p. 45.—*C. Aurelianus*, Acut. morb. Lib. III. C. 17.—*Celsus*, Lib. IV. C. 13.—*Galen*, De loc. affect. Lib. VI. C. 2. Definit. med. 273, De sympt. caus. Lib. I.; and others.

[2] Ed. Kühn, T. II. 507, 509; T. I. 182.

The essence of ileus (if we consider only those cases in which the symptoms of occlusion of the intestine are presented) was supposed, by all the ancients, to lie in an inflammation of the bowels,[3] which was attributed by Hippocrates especially to flatus, but by Galen, likewise, to collecta materia (pituita) in intestinis, to abscessus in substantia intestinorum, to obstructio stercoris aridi, and to "scirrhus." The ileus herniosus of strangulated hernia was also thought to be due only to inflammation of the contents of the hernial sac, set up by the gases or fæcal matter. According to Galen, another result of the inflammation is seen either in arrest of peristaltic action and absolute constipation, or in reversal of the peristaltic action with vomiting of fæcal matter.[4]

During the Middle Ages, and for a long time thereafter, the theory of ileus, both with regard to the application of the term to various painful abdominal affections, and so far as the knowledge of its nature was concerned, remained as taught by Hippocrates and Galen. The Arabians added nothing to it beyond a division, —as arbitrary as it was unnatural—of ileus into five degrees. Paracelsus and van Helmont presented new views of its nature. The former saw its cause in a contraction of the intestine, caused by "the irritation of increased tartarus," the latter in an increased formation of gas. In this way they both added to the obscurity. Afterwards the discussion was mainly as to what was the most important symptom of ileus. Some thought it was the pain, and chose the name "dolor iliacus;" others thought it was the vomiting, and named it "vomitus iliacus;" others found it in the distention of the abdomen, and called it "tympanites." Many drew distinctions, as did the old Greek physicians, according to the seat of the pains; and we find descriptions of this affection in their writings, sometimes under the head of "passio iliaca" or "colica," sometimes under that of "malum hypochondriacum."

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[1] Εἰλεός, εἴλημα (Hippocrates), εἰλεόν (Aretæus), from εἰλέω, εἴλω=concludo, coarcto, or from εἰλέω, εἰλύω=torqueo, volvo. If the pain was above the navel, the disease was called εἰλεός; if about it, στρόφος; if below it, κόλικος. The word chordapsus had different meanings even among the ancients. χορδή is used for intestine; ἅπτω=necto is also used with the signification of accendere.

[2] *Aretæus*, l. c.; *Celsus*, l. c.; *Galen*, De loc. affect. Edit. Kühn. Vol. VIII.; and others.

[3] "Resiccatur enim intestinum et constipatur ex inflammatione ita ut neque flatum neque alimenta transmittat. Venter durus fit et vomit interdum imprimis quidem pituitosa, deinde vero biliosa, tandem etiam stercus. . . . . Volvulorum autem caussam a flatibus dependere cuivis esse manifestum existimo. Horum enim spiritus trajectio caussa est." *Hippocrates.* Edit. Kühn, II. 304. I. 578.—"Volvulus est phlegmone intestinorum quo malo nec flatus infra, nec dejectiones transmittuntur, tormina sequuntur cruciatusque intolerabiles."—*Galen*, Definit. med. CCLXXIII.

[4] Edit. Kühn, T. VII. 220; T. XVIII. A. p. 68.

The knowledge of the anatomical lesions underlying ileus increased gradually when autopsical examinations began to be made more frequently and carefully under the influence of the start taken by anatomy in the sixteenth century. A close examination of the anatomical literature of those times shows that even in the seventeenth, and at all events in the middle of the last century, most kinds of occlusions and constrictions of the intestines were well known to the anatomists. Realdus Columbus, Vesalius's prosector, Fab. Hildanus, and Riolan in the sixteenth, Peyer and Ruysch in the seventeenth century, described invagination of the intestine; Fabric. Hildanus and Riolan, Tulpius, Bonnet and Boerhaave in the seventeenth century made the profession acquainted with ileus due to compression of the intestine by different abdominal tumors. Alberti in the sixteenth, Bonnet in the seventeenth, De la Peyronie in the beginning and Monro in the middle of the eighteenth century, told of constriction of the intestine by false ligaments and omental bands; Felix Plater in the sixteenth, Blasius and Barbette in the seventeenth, Burserius in the eighteenth century, described twisting; Riverius in the sixteenth, Bonnet and Bartholinus in the seventeenth, Burserius in the eighteenth century described the formation of knots in the intestine and "conglomerations;" Haller, Fabric. Hildanus, Petrus Salius Diversus published cases of ileus caused by carcinoma and scirrhus of the intestinal canal; Paræus, Fab. Hildanus, Ballonius in the sixteenth, Blankard, Sennert, Schenk, Riverius, Blegny in the seventeenth century, described diaphragmatic hernia. Cases of congenital occlusion of the anus were mentioned in the seventh century by Paulus of Ægina, in the fifteenth by Benivenius, in the sixteenth by Mercurialis, Fab. ab Aquapendente, Donatus, and others. Cases of occlusion by gall-stones and by intestinal stones were communicated by Malpighi and Ruysch in the seventeenth, by Chaptal and Sandifort in the eighteenth century, while Fernelius in the sixteenth, Kerckringius and others told of occlusion by foreign bodies. Diverticles in the canal were first mentioned by Buonazolius and Fabr. Hildanus in the sixteenth, were frequently and fully described by Ruysch in the seventeenth and Littre in the eighteenth century, and finally their mode of formation was explained by Meckel. Descriptions of constriction by a diverticle are first found in the middle of the eighteenth century (Moscati, Van Doeveren), and about the same time hernia duodenojejunalis was first described by Neubauer, and strangulation of the intestine in abnormal openings in the mesentery and omentum by Brambilla, Monro, Callisen, and De Haën. We have also to mention the numerous examples of the most varied kinds of occlusion and constriction of the intestine which are contained in the great anatomical compilations of the seventeenth century made by Bonetus, Spigelius, Fantonus, Bartholinus, Tulpius, Manget, Ruysch, and also in the later works by Lieutaud, Sandifort, Baillie, and others.

Although, as has thus been shown, the anatomical manifold lesions underlying ileus were well known to the anatomists of the sixteenth and seventeenth centuries, yet we should greatly err were we to believe that their knowledge at once transformed the ideas held by the physicians. We cannot easily form a better conception of the indifference with which the physicians of that time, accustomed to purely symptomatic notions of disease, received the anatomical discoveries than by comparing the condition of the anatomists' knowledge of the causes of ileus with the opinions held and taught by contemporaneous medical authorities. Leaving out the ileus inflammatorius, ileus a stercore arido and a skirrho of Hippocrates and Galen, dynamic ileus plays everywhere the principal part in etiology, produced by a change of the humores acres mordentes, biliosi, pituitosi (ileus humoralis of Sennert, Cartesius), or of a materia febrilis (Sydenham), or of gases (ileus tympanicus, flatulentus, physodes). Even Fr. Hoffmann, at the beginning of the last century, spoke of ileus as arising "a mordentibus succis biliosis, a sanguine in vasis tunicarum intestinalium hærente," and "a caussa calida." Sydenham knew "mechanical ileus" due to occlusion of the bowel only as an "ileus nothus" allied to the real dynamic ileus. Nor were there few who held the anatomico-pathological processes to be the indifferent consequences of the ileus, the products of the motus antiperistalticus, and even those to whom the significance of the mechanical occlusion of the intestine in the symptomatology of ileus was evident, considered the anatomical facts rather as incidental, secondary ones, and sought for their cause, according to the ruling notions, either in a primary inflammation, or in the reversal of the peristaltic action, and in a change of the juices, or in an abnormal formation of gas. Thus cause and effect, primary and secondary, were mistaken and interchanged. The adherents of ileus inflammatorius saw in the peritonitis, which was almost always found at the autopsy, a justification of their view that inflammation was the primary cause of ileus. Knotting of the intestine and internal strangulation were interpreted as the result of motus intestinorum inversus, which, in its turn, was due to the displacement of strong juices or to materia febrilis or to gases. The contracted condition of the intestine below the occluded point, or the unimportant contractions occurring in the colon during the death-struggle, served the adherents of ileus spasmodicus as a proof of the correctness of their opinion of the spasmodic nature of the disease, and the believers in ileus pituitosus enjoyed a supposed triumph when Fernelius[1] found a colloid cancer of the intestine and called it "obstruction of the bowel by mucus." The anatomist Ruysch characterized this difference in the views of ileus held by anatomists and physicians when he closed a convincing account of the anatomical causes of intestinal obstruction with the words: "Qualis autem sit affectus ileos, quaeque proprie ejusdem caussa haud satis convenit inter medicos."

Since occlusion of the intestine leads to distention of the abdomen by gas, this latter was thought to be the essential part of ileus, and the finest hypotheses concerning the formation of the gases as "ileus excitors" were imagined. This lasted a long time, until light was thrown upon the location of the gases. Boerhaave (Aphor. 1226, 1251) even believed that they "inter membranas et in dilatatis glandularum cavis hærere;" others supposed that they were to be found normally as well without as within the intestines, and passed freely through the pores of the walls ("aër foras exit reditque"), that occlusion of the pores caused confinement of the gases and thus gave rise to ileus.[1] Gorter, in the eighteenth century, believed that the mesentery was the seat of the formation of gas in ileus, an opinion which had been previously advanced in the sixteenth century by Riverius and Forestus, although Duret (1550) had distinctly declared that the gases were formed within the intestine, and their presence outside was merely the result of a perforation.

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[1] *Bonnet,* Sepulch. Anat. Sect. XIV. Obs. 25.

In consequence of the appearance of stercoraceous vomiting, Galen (l. c.), and afterwards, in the sixteenth century, Jacobus Sylvius[2] especially, explained ileus as due to a "reversal of the normal movements of the intestine." It was thought that this motus peristalticus inversus seu praeter naturam ordinarily occurred without any mechanical obstruction in the canal, and was produced by inflammation, or by displaced juices or gases. The existence of such an ileus peristalticus, without any change in the permeability, was thought to be proved by the "observation" related by Galen and believed, most singularly, by the best authors of the later periods (Dolaeus, Sennert, Diemerbröck, Sydenham, van Swieten), that in cases of ileus suppositories and enemata, introduced into the rectum, were afterwards vomited by the mouth—observations which, as Boerhaave assures us, passed "testibus gravissimis viris" for unquestionable facts, and were truly narrated by the writers.

Intestinal worms played an important etiological part. As there was almost no other affection of the intestines, and, we may say, no disease whatever, of which these, among other things, were not supposed to be the cause, so was it also with ileus. From the time when Gordon, in the fourteenth century, presented volvulus verminosus, based upon the vomiting of an ascaris during an attack of ileus, until the present century, this classification was held in undiminished respect, even by the best authorities. New confirmations of the influence of ascarides in producing ileus were furnished afterwards by autopsies, in which they are often, and indeed from allied causes, found in the neighborhood of the occluded point, so that an apparently well-grounded suspicion must fall upon them. The adherents of spasmodic ileus saw in these worms the exciting cause of a convulsive contraction of the intestine leading to ileus. Many representations of conglomerations of worms have been handed down to us from former times, which were thought to have caused fatal ileus by total obstruction of the bowel. It is scarcely to be doubted that in such cases, after the bunch of worms had been found, care was not taken to seek further for the anatomical cause of the occlusion of the intestine.

The nerve-pathologists, finally (Cullen, Pinel, Alibert), set up against this ileus inflammatorius an ileus nervosus, which, according to them, was sometimes spasmodicus, sometimes paralyticus, sometimes antiperistalticus. This was explained by a supposed convulsive or paralytic condition of the intestine, due to abnormal nerve-action, a reversal of the peristaltic motion by "pathological innervation." The idea of an ileus spasmodicus, like an icterus spasmodicus, lasted the longest, even to the middle of the present century. To-day the question of the existence of such an affection no longer calls for serious discussion.

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[1] Hence the popular "cure" of ileus by means of dry cups, which were thought to suck up the gases and restore the permeability of the pores of the intestine.

[2] J. Sylvii Ambriani Med. Op. med. Genev. 1630. Definitio ilei; "Expultrix intestinorum facultas depravata et sursum pellens vel abolita."

Tardily and gradually, as under the influence of the anatomico-pathological direction given to medicine, the field of ingenious hypotheses, of traditional and preconceived opinions was abandoned, and that of simple observation was again occupied; and as a healthy logical need made itself felt, the need of bringing, in the simplest way, and without the aid of obscure hypothesis, clinical observations into agreement with the lesions found upon the cadaver, more and more light was thrown upon the nature of ileus. Men became convinced of the variety of the anatomical processes which had previously been comprised under the indefinite term ileus; they recognized that different anatomical facts presented similar, or even identical symptoms during life and in the course of the disease, and often could not be distinguished clinically from one another. The need developed at the same time of anatomical differential diagnoses led to a much sharper distinction and more careful weighing of the symptoms, of the beginning, and of the course of the disease. The symptomatic generic idea ileus was broken up into a series of anatomical diagnoses, such as peritonitis, typhlitis, coprostasis, occlusion, and constriction of the intestine, each of which again presented new questions of detail, both anatomical and etiological. But now the traditional idea of ileus, instead of being dropped, was still continued. Some designated by this word those grave symptoms which belong to occlusion of the intestine; others, starting from the fact that of all the symptoms of occlusion there is only one, stercoraceous vomiting, which positively proves the loss of permeability of the canal (except when it is due to the presence of a fistulous communication between the stomach and colon, or when it occurs in coprophagic lunatics), used the expression ileus, without regard to its etymological origin, as synonymous with stercoraceous vomiting, and spoke of ileus only when the other was present (J. Frank, Schoenlein [1]). Even if no real objection can be made to this traditional and arbitrary use of the word ileus, yet it is plain that it lays too much stress upon a single symptom, stercoraceous vomiting, which, moreover, is often absent in these same cases of occlusion, when, for example, the course of the disease is very rapid, or when the occlusion is seated high up in the small intestine. To-day, when we are no longer satisfied with symptomatic diagnosis, but make the distinctions according to anatomical or etiological criterions, the idea ileus stands upon the same footing as hæmoptysis or hæmatemesis. Our diagnosis tries, even during life, to be anatomical; it seeks to make out by the aid of objective symptoms and the history of the case, not only the condition of the interrupted or diminished permeability of the intestine, but also the location, the cause, and the nature of the obstruction.

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[1] "Morbus quo stercora parum, non satis aut omnino non per anum, contra per vomitum simul cum doloribus et anxietate redduntur ileus dicitur." "Ileus is the set of symptoms of occlusion of the intestine ending in stercoraceous vomiting."

Classification.

Constrictions and occlusions of the intestines are either *congenital* or *acquired.*

Among the congenital occlusions, that of the anus or rectum is the most frequent. It consists either of an agenesia ani, wherein the formation of the anus is not completed, or in an atresia ani, in which the anal cul-de-sac is formed, but has not united with the inferior blind end of the rectum. The distance between these two culs-de-sac varies from the thickness of a simple membranous septum to that consequent upon complete absence of the rectum, in which latter case the colon usually ends as a blind projection. With both kinds of congenital occlusion there is found, in about forty per cent. of the cases (my statistics), an abnormal opening of the rectum into the bladder, urethra, or vagina (recto-vesical, urethral, vaginal fistula). The proportion of the sexes, according to my statistics of 375 cases, is: 241 males, 134 females. If we take Zöhrer's records of the lying-in hospital at Vienna, and Collins's of the lying-in hospital at Dublin, we find that in 66,654 new-born children, the anomaly in question was observed three times.

Congenital atresia occurs less frequently in the colon and small intestine than at the anus and in the rectum. For 375 cases of ano-rectal atresia, we find 10 of the colon and 74 of the small intestine. Without entering into the details of my statistics—which are reserved for another occasion—I must yet briefly mention here some of the more important facts. 1st, Congenital atresia of the colon occupies, almost without exception, the sigmoid flexure. It is generally the result of a fœtal mesenterial peritonitis, which has led to constriction and wasting, associated, as is frequently the case, with twisting of the sigmoid flexure about its mesentery. 2d, Atresia of the small intestine occurs preferably at certain points. *a.* In the duodenum these are most often the point where the bile and pancreatic ducts open, and the point where the duodenum becomes the jejunum under the transverse mesocolon. The atresia or stenosis which occurs here is very often of the valvular kind--that is, it is formed by a membranous septum which looks exactly like an abnormally large valvula connivens, or as if two of them had united. *b.* In the ileum the upper and middle portions are very rarely the seat of atresia; it is found frequently at the ileo-cæcal opening itself, or, still more often, from one to three inches above it at the point where the ductus omphalo-mesentericus is given off. A twist, due to the twisting of the umbilical cord, exists normally at this point, and is important in determining the position of the intestine.[1] If this extends abnormally to the intestine itself, of which the ductus is only a continuation, atresia occurs at the point where the latter is given off in consequence of the twisting.

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[1] *Kölliker*, Entwicklungsgesch. S. 360—363.

Other atresiæ of the small intestine depend upon similar anomalous developmental incidents, and upon the rather complicated disposition of the intestinal tract of the embryo. Fœtal peritonitis, in particular, plays an important part. It not seldom leads to multiple atresiæ and stenoses, as many as ten of which have been found in the small intestine (Schüppel). Congenital atresia of the small intestine is as frequent in one sex as in the other. For the other details the reader is referred to the accounts, generally inadequate, given in the surgical manuals and in text-books, as well as to the following bibliography.[1]

We also distinguish intestinal constrictions and occlusions according to the *anatomical causes.*

1. Causes which obliterate the lumen of the canal by pressure from without—*compression* in the broader meaning of the word.

Here belong strangulation (incarceration) of the intestine in holes, fissures, and hernial rings; strangulation by false ligaments, the appendix vermiformis, and the diverticle; compression by tumors, by the mesentery, by dislodged viscera. We include here also twisting (torsion) and formation of knots; for in these, too, the occlusion is due to the pressure which the adjoining parts exert upon each other.

2. Causes which obstruct the lumen of the intestine—*obturations:*

a. Causes which are not continuous with the wall of the canal: gall-stones, intestinal stones, foreign bodies, fæcal masses.

b. Causes which are continuous with the wall of the canal, expand within it, and obstruct it: neoplasms, especially those shaped like polyps.

c. Obstruction by the bowel itself: intussusceptions.

3. Causes which, starting from or encroaching upon the wall,

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[1] Besides the anatomico-pathological manuals and atlases of *Meckel*, *Cruveilhier*, *Ammon*, *Vrolik*, *Förster*, *Albers*, and others, must be mentioned especially: *Schäfer*. De canali intestinali a prima conformatione, etc. Würzburg. 1825.—*J. Frank*, Prax. med. P. III. Vol. II. Sect. I. p. 18.—*Amussat*, Mém. sur la possibilité d'établir, etc. Paris. 1843.—*Curling*, Med. chir. trans. XLIII.—*Idem*, Dis. of rectum, trans. by Neufville. Erl. 1853.—*Thüngel*, Ueber künstl. Afterbildung. Kiel. 1853. Hirschsprung. Copenhagen. 1861, and in Schmidt's Jahrbuch. Bd. 117, 310.—*Günther*, Lehre von der blutig. Operat. Part IV.—*Ashton*, Diseases, etc., of the rectum, trans. by Uterhardt. Würzburg. 1863.—*Goyrand*, Gaz. méd. de Paris. 33-41. 1856.—*Friedberg*, Arch. gén. de méd. 1857. V. 9.—*Friedberg*, Virch. Archiv. XVII. 1859.—*Schleiss*, Henle and Pfeufer's Zeitschr. N. F. III. 3. 1853.—*Fiedler*, Arch. d. Heilk. V. I. 1864.—*Schüppel*, ibid. V, 78.—*Küttner*, Virch. Arch. Bd. 54, 1872.

narrow the lumen of the intestine either circularly or by kinks and distortions.

Here belong the constrictions, distortions, and angular obstructions of the canal caused by chronic peritonitis, cicatricial stenoses, and neoplasms, which produce the narrowing sometimes by infiltration of the wall, sometimes by constriction.

Finally, we distinguish clinically between the grave phenomena of *total definitive occlusion* and the symptoms of *partially interrupted permeability.* The latter offers all the possible degrees between simple constipation due to functional causes and the more important disturbances of permeability. In the severest cases the clinical appearance is that of repeated temporary occlusions, and is more marked as the advance of the contents of the intestine is interrupted more frequently and for longer periods.

If we examine the above-mentioned anatomical causes from the clinical standpoint, we find that the different anatomical processes of constriction and occlusion of the bowels sometimes differ essentially in their clinical history, and sometimes resemble one another exactly ; but still we must make the following general division :

a. Causes which *appear acutely*, not seldom suddenly, *and lead at once to total definitive occlusion of the intestine.*

Among these belong the anatomical causes mentioned in the first section—incarceration, strangulation, twisting, and formation of knots, acute compression of the intestine—by the mesentery, for example—etc. Many gall-stone obstructions act in the same way; intussusception rarely takes this course—mainly in cases of invagination of the ileum in children.

b. Causes which generally *appear acutely*, *produce notable disturbances of permeability*, but not immediate total occlusion, and, if recovery does not take place, lead, sooner or later, to definitive occlusion, or to death in some other way.

In this way intussusception generally acts, as do also incomplete incarceration and acute incomplete compression, and, lastly, many of the obstructions caused by gall-stones and foreign bodies.

c. Causes which *arise slowly and excite*, for what is ordinarily a long time, *symptoms of disturbed perviousness of the* *intestinal canal*, until they lead, either suddenly and once for all, to definitive occlusion, or, without this, to a fatal termination through peritonitis, perforation, or in some other way.

Here belong the anatomical causes, mentioned in the third section, of narrowing of the canal by cicatricial bands, neoplasms, chronic peritonitis; also compression by gradually-increasing tumors, most cases of obstruction by intestinal stones, and, lastly, chronic fæcal obstruction dependent upon functional causes.

We distinguish, in constrictions and occlusions of the intestine, a number of symptoms which depend solely upon the mechanical fact of occlusion of the canal, and are, therefore, the same, however the anatomical process may differ in the individual cases. This group of symptoms is modified only by the location and degree of the obstruction. A set of other symptoms, and especially certain peculiarities in the course, duration, and prognosis, depend upon the special anatomical cause of the occlusion. From this point of view, which is especially important in diagnosis, a closer study of the pathology of the different anatomical causes of constriction and occlusion is necessary. If we should give a complete pathological account of each of these causes, frequent repetitions would be unavoidable, and I prefer to deviate from the usual plan of treating the subject to this extent, that I shall consider in turn:

1. *The anatomico-pathological changes in general*—that is, those which are found upon the cadaver under different conditions of occlusion and constriction of the intestine.
2. *The pathology of occlusion and constriction of the intestine in general.*
3. *The different kinds of occlusion and constriction of the intestine, with their anatomical and clinical peculiarities.*
4. *Treatment.*

Anatomico-Pathological Changes in General.

These interest the intestines and the cavity of the abdomen. The portion of the intestine which lies above the occluded point differs essentially in size and appearance from that lying below it. While the latter appears empty and much contracted (contraction of inanition), the former is filled and distended partly by gas, partly by a considerable quantity of liquid feculent matter, a distention which extends from the seat of the occlusion upwards through the whole canal, and not seldom affects the stomach also. The latter organ, especially when the occlusion is located in the duodenum or jejunum, often presents a high degree of ectasia, except when the course of the disease has been very acute. The colon above a chronic narrowing of its lower portion often becomes longer, and consequently more convoluted.

In cases of acute, rapidly fatal occlusion of the colon, for example when twisting of the sigmoid flexure has occurred, the distention has been repeatedly found limited to the colon and cæcum, the latter especially being enormously distended.[1] This can only mean that in such hyperacute cases the ileo-cæcal valve was sufficient, and prevented the reflux into the ileum of the obstructed contents of the colon, while the contents of the small intestine, pressed forward by a powerful peristaltic action, were still able to overcome the increased pressure in the cæcum and to fill the latter to excess. But soon, and the more promptly the nearer the occluded portion of the colon is to the ileo-cæcal opening, the valve becomes insufficient under such conditions, and the distention spreads, even when the occlusion occupies the rectum, over the whole of the small intestine.

The peritoneum is found in different conditions. In hyperacute cases of internal strangulation, for example in infantile invagination of the ileum with a rapidly fatal course,[2] there is sometimes found, together with an intense hyperæmic injection of the blood-vessels, and a loss of polish on the surface of the serous membrane, only an inconsiderable peritonitis[3] limited to the seat of the occlusion. In other, and indeed in most, cases the peritoneum is the seat of an inflammation as intense as it is widespread, which has led to numerous adhesions of the distended intestinal loops, to sero-fibrinous, fibrino-purulent, or ichorous exudations in the dependent parts of the abdominal cavity and between the adhesions. Not seldom after separation of the loosely adherent convolutions the places at which they were brought into reciprocal contact are marked by red, vascular, raised, fibrinous lines of exudation, while the surfaces of contact of the adherent loops often offer an almost unchanged aspect.

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[1] *E. g.*, Fagge, l. c. Case 22. p. 306.

[2] Prag. Vierteljahr. Bd. 121. S. 25.

[3] For the more minute processes, see Peritonitis in a later volume.

The peritoneal exudation is sometimes hemorrhagic, and even large quantities of blood are found in the abdominal cavity as well as within the intestines.[1] This is the case when the nature of the occlusion is such that large or many small veins of the mesentery are compressed, the flow of blood through them checked, and hemorrhage by diapedesis induced, thus, for example, in extensive invaginations, in cases of twisting and knotting, in strangulation of large loops. The abdominal walls often appear infiltrated with blood in different degrees, sometimes in numerous ecchymotic spots, sometimes in larger strips of uniform color varying from a light to a dark red (apoplexia intestini), alterations which usually increase in intensity as they approach the seat of the obstruction, where the intestine is often covered with a layer of black clotted blood, and its walls are black, friable as tinder, and gangrenous.

The hemorrhagic infiltration of the intestinal mucous membrane, often extending high above the obstruction, leads occasionally to diphtheria of the mucosa. This appears in the form of parallel, dirty-gray streaks, corresponding to the height of the folds of the mucous membrane, and raised from the dark, ecchymotic mucosa. The gangrenous destruction, sphacelus of the wall of the intestine often leads to perforation. The escape of fæcal gases when the abdomen is opened, the finely divided particles of fæcal matter adhering to the peritoneum and mingled with the liquid exudations, mark peritonitis due to perforation.

It is often difficult to find the perforation upon the cadaver, both because the distended bowel collapses and the opening closes again, and because during the search, even when it is carefully conducted, new perforations are made in the friable, tinder-like walls. Occasionally the perforation takes place at the moment the abdomen is opened. When the distended intestines are suddenly freed from the confinement of the abdominal walls and crowded forcibly forwards, the rotten, strangulated parts tear, the sound of the escaping gas is heard, and at the same time the sinking in of the projecting intestines demonstrates their relief by perforation. The autopsical examination of internal incarcerations is a most difficult task, and a skilful as well as a careful hand is needed for the clear demonstration of the often complicated relations. The indications of gangrene and hemorrhagic infiltration of the intestinal walls are difficult to distinguish from the cadaveric processes of decay and imbibition of the tissues with blood, a circumstance which makes it desirable that the examination should be promptly made.

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[1] Interesting cases of this kind are given by Küttner. Virch. Arch. Bd. 43.

Other changes, differing from the gangrenous ones seen at the occluded point, are found spread over a certain extent above it. They consist of more or less deep sloughs of the mucous membrane and the consequent ulcers, and they are due to the partly mechanical, partly chemical action upon this membrane of long retained and altered fæcal masses. Such so-called *stercoral ulcers due to gangrene by pressure* are found most frequently in the cæcum and colon, in cases of constriction of the latter. Sometimes they pass beyond the ileo-cæcal valve, and extend into the lower portion of the ileum. In the cæcum they often run together and form large ulcers. They may give rise to perforation.

Perforation of the intestine can be prevented by adhesions with the neighboring organs or with the omentum. If it occurs with simultaneous encapsulation, by means of false membranes, fæcal cloacæ or sacculated fæcal abscesses are formed; if it opens into the retroperitoneal cellular tissue an ichorous phlegmon is set up. A rare result is the spontaneous opening outward through the ulcerated walls of the abdomen of the intestine which had previously become adherent thereto (fistula stercoralis, anus praeter naturam); another, still rarer termination, is the opening of the portion of intestine which lies above the occlusion into that which lies below it; this may take place directly when the two loops have become united by adhesions (fistula bimucosa), or through a fæcal cloaca, or even through a longer fæcal canal. Perforation into other cavities sometimes takes place, especially in cases of constriction of the rectum, as, for example, into the vagina, and exceptionally even into the bladder.

From the increased intensity and frequency of the peristaltic efforts with which the intestine above the constriction struggles against the obstacle, there often results a rather notable hypertrophy of its muscular coat; the chronic catarrh and hyperæmia lead to thickening of its mucous membrane also. This enlargement of the walls must be considered to be all the greater, as dilatation exists at the same time—a condition which we may very properly term excentric hypertrophy of the intestine. This is a real hypertrophy, due to work. It undertakes, like certain portions of the heart in cases of stenosis of its valves, to compensate for the narrowing of the intestinal canal; and, as in the heart, so here also, serious disturbances of compensation follow degeneration of the hypertrophied muscle, which may lead to arrest of the contents and to total impermeability.

The other lesions which we find in the bodies of those who have died in consequence of occlusion of the intestines, differ according to the course, duration, and particular kind of the affection. We have to mention the extreme emaciation of the body in cases of stenosis, especially when due to malignant growths, leading to death by inanition, the appearance of the body and the condition of the individual organs often resembling that seen in cholera (dryness of the connective tissue, of the serous membranes, of the muscles, the bluish-gray color of the skin, contraction of the bladder, etc., etc.), in acute, rapidly-fatal cases of incarceration. Among the lesions of the different organs, we often find elevation of the diaphragm in consequence of the meteorism, hypostases in the lungs, venous hyperæmia and œdema of the skin, increase of cerebral liquid, etc. When fæcal infiltration and sloughing of the retro-peritoneal connective tissue have taken place, we sometimes have infectious emboli, pulmonary infarctions, and embolic pneumonia, ending in abscess or gangrene.

We have yet to mention briefly a lesion which is found in the lungs of those who have succumbed to ileus. I refer to pneumonia caused by the presence of foreign bodies in the lungs, particles of vomited matter (fæcal matter when the vomiting is stercoraceous) carried in during inspiration. This can occur the more easily, as the patients are often rendered comatose by large doses of opium, and the reflex irritability of the mucous membrane of the respiratory passages is diminished. Like most pneumonias of this kind, they occupy mainly the lower or middle lobe of the right lung, where infiltrations (sometimes distinct lobular ones, sometimes united and forming lobar ones) are found with a dirty-gray or dark-green surface of section, central sphacelated softening, and formation of gangrenous cavities filled with offensive matter. The surrounding lung tissue is more rarely in a condition of hepatizing inflammation than in that of soft, moist infiltration. Microscopical examination of the gangrenous spot clearly shows, as I have occasionally demonstrated, that aspiration of the contents of the intestines has taken place, for we find there pieces of muscle stained with bile, bits of vegetables, grains of starch, etc.[1]

Pathology of Occlusions of the Intestines.

Symptoms.

Let us take a case of acute internal strangulation for the basis of our picture. Either suddenly, during perfect health, or preceded by an insignificant diarrhœa or constipation, perhaps after some incidental cause (dietary error, purge, injury, chilling), violent, rapidly-increasing colicky pains appear, which sometimes spread over the whole abdomen, and sometimes are more circumscribed, and limited especially to the region of the umbilicus. The pains, which offer remissions and exacerbations at short intervals, and are sometimes convulsive or parturient-like, resist the domestic remedies generally employed before the arrival of the physician, and are often combined with an unusual physical and mental excitement of the patient. The latter and the physician himself suppose it to be a case of unusually violent colic, due, perhaps, to fæcal obstruction, and expect relief through a free passage from the bowels. But success does not follow the use of the milder purgatives, nor of the more powerful or drastic ones prescribed, unfortunately, only too often, notwithstanding the violent pain and even the vomiting. Except for a few, scanty fæcal masses voided by the aid of injections, the abdomen remains closed. Soon after the appearance of the pains, sometimes excited by the purgatives that have been administered, eructations occur, followed by vomiting, repeated at diminishing intervals. The abdomen becomes distended and tympanitic ; the features, at first drawn by the pain and anxiety, contract, as in the well-known facies cholerica ; the extremities become cold ; prominent points livid ; respiration superficial, frequent, and of the supra-costal type ; the pulse small and frequent. A painful desire to empty the bowels is kept up and increased by the failure of all attempts to purge, by the absence of flatus, and by the sense of oppression produced by the meteorism. The vomited matter, composed, first, of the contents of the stomach, and then of quantities of green bilious matter, becomes visibly discolored, dirty-green, then brownish, and presents more and more the aspect of diarrhœal passages, until, finally, fæculent masses, preceded by fæcal ructus, are vomited (ileus, miserere). At this time the patient, in a condition of extreme collapse, covered with cold sweat, tormented by frequent vomiting, singultus, dyspnœa, colics, and unquenchable thirst, with hollow eyes set in bluish rings, pointed nose, sharply defined features, with leaden, faintly-livid complexion, with muffled voice and dirty-brown, dry tongue, presents a horrible picture, intensified by the fact that the mental functions are generally undisturbed.

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[1] Compare *Buhl*, Zwölf Briefe über Lungenentzündung, etc. München. 1872. S. 19.

Let us now analyze the individual symptoms and their modifications caused by the seat of the occlusion.

The pain is caused partly by the rupture of the peritoneum at the strangulated point (the effect of the constriction), partly by the meteoristic condition, distention of the intestinal walls, partly by peritonitis.

The periodical exacerbations of the pain are due to the periodically-increased peristaltic pressure against the obstruction, and to the compression exerted thereby upon the nerves of the hyperæmic or inflamed peritoneum.

The intensity of the pain depends upon the excitability of the individual, upon the condition of the sensorium, the state of the peritoneum, and especially upon the extent of the peritonitis. Pains which are produced or increased by slight pressure upon the abdomen indicate the existence of peritonitis. Sometimes, while the sensorium is still intact, the pain and vomiting cease quite suddenly a short time before death, and the meteorism and collapse increase. This proceeds from paralysis of the intestine above the obstruction, from the arrest of the peristaltic action, which in part occasioned the pain as well as the vomiting; or it is, as is often ascertained, the immediate result of the occurrence of perforation.

Vomiting sometimes appears at the beginning, simultaneously with the pain. This initial vomiting is the result of the shock of the strangulation, like that which we see in the shock following injury to the abdomen. The irritation of the peritoneum produced thereby excites by reflex action the central ends of the nerves which supply the muscles thrown into action during the act of vomiting, the diaphragm and the abdominal muscles. Another vomiting comes later, and is partly the result of the inflammatory irritation of the peritoneum, partly that of the arrest of the contents of the intestines. Both factors act as irritants, and excite not only vigorous contractions of the bowels, but vomiting also. Vomiting due to arrest of the contents naturally appears the earlier, and is the more violent, the higher the situation of the obstruction; it also appears sooner when peritonitis sets in earlier, and when the patient is more irritable (hence it is especially early in children), or when the intestinal canal has been irritated by the administration of drastic purgatives or similar drugs. When the obstruction occupies the uppermost part of the small intestine (the duodenum below the orifice of the ductus choledochus, jejunum), the vomited matter is always deeply colored with bile; but long retention of the obstructed masses, in consequence of the use of opium, for example, can discolor them and cause them to smell foully, and even to be mistaken for stercoraceous matter, as happened in one of Cruveilhier's cases of occlusion of the jejunum. When the occlusion is at the middle of the ileum, the vomiting, under the same circumstances, often assumes a fæcal-like character; when it is in the lowest part of the ileum or in the colon, stercoraceous vomiting occurs. This consists of thin liquid, feculent masses. It is only very seldom that in cases of occlusion of the colon larger pieces of fæces are vomited, for the firmer fæcal lumps of the colon are finely broken up, and dissociated by their passage through the ileum filled with liquid.

The erroneous assertion has been repeatedly made, that stercoraceous vomiting can occur only when the occlusion is situated in the colon, although it is easy to prove by autopsical examination that the contents of the lower portion of the ileum often bear the character of soft, sometimes even harder fæces, a fact which De Haën[1] has already opposed to the above-mentioned opinion. On the other hand, it has been said that stercoraceous vomiting, depending upon the passage of fæces from the colon into the ileum, was not possible, because the ileo-cæcal valve prevents the retrograde movement of the contents of the intestine. It was believed that the contents of the colon could be vomited only when fistulous communication existed between the colon and the stomach. But the ileo-cæcal valve does not form a permanent obstacle; it becomes insufficient, as we have already explained, in consequence of great distention of the colon or cæcum and simultaneous paralysis of the cæcal sphincter.[1]

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[1] l. c. P. II. p. 89. Also *Stoll*, Ratio med. T. 1. p. 237.

The explanation of the mechanism of stercoraceous vomiting has called forth different opinions. Formerly it was thought to furnish the strongest proof of the existence of an antiperistaltic motion, and an "ileus verus" was imagined (Sydenham) which was based solely upon the reversal of the peristaltic motion. The question of the occurrence of antiperistaltic action has been settled, so far as the stomach is concerned, for it has repeatedly been the subject of observation during vomiting, but it has not yet been decided for the intestine. The intestinal movements that can be seen after the abdomen is opened, as can be easily demonstrated by experiments upon animals, are very irregular. Contractions and indentations beginning here and there, oscillatory and rolling movements, and when a wave has passed regularly over a certain portion of the intestine, it is often very difficult to say whether it pased from above downwards or in the opposite direction. Betz,[2] Schwarzenberg,[3] and Houckgeest,[4] could not convince themselves of the existence of antiperistaltic movements, either under normal conditions or after the application of a ligature about any part of the canal. On the other hand, Engelmann[5] saw two waves start from the irritated point on the intestine—one peristaltic, the other antiperistaltic. I shall not discuss these opposing theories. The mechanism of stercoraceous vomiting does not necessarily require for its explanation the existence of antiperistaltic intestinal movements. Whether these do or do not occur, the main fact is always another and a very simple one, one brought forward by van Swieten,[6] and, since Henle, almost universally accepted. The intestines above the obstruction become more and more filled up by their accumulating contents, and finally to such a degree that every attempt to vomit, every contraction of the abdominal muscles, of the diaphragm, and, if you choose, of the intestines also, moves the contents in the only direction in which they can go—that is, upwards from the point of occlusion. It is evident that under these circumstances the contents would be moved by normal peristaltic in the same direction as by antiperistaltic action.[7] On the other hand, in favor of the antiperistaltic action we have: 1st. Those very rare cases in which stercoraceous vomiting is observed in the course of a diffuse peritonitis without demonstrable alteration of the permeability of the intestines (compare the details given in the chapter on Ileus Paralyticus). 2d. The fact that in cases of acute internal incarceration the stercoraceous vomiting occurs only a few hours after the beginning of the attack, at a time when it cannot well be thought that there is any great accumulation of intestinal matter (see the transudation hypothesis to be hereafter described). 3d. The movements of the intestines, in cases of inflammation and other irritation of the peritoneum, differ, so far as their quality and effects are concerned, from what is normal. This is shown by the circumstance that, for example, in cases of acute peritonitis, notwithstanding the increased vigor of the movements (which can often be made out with certainty by auscultation of the abdomen), no diarrhœa appears.

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[1] For details see the section on treatment.

[2] Württemberg Corresp.-Bl. 1850.

[3] Zeitschr. f. rat. Med. VII. 3.

[4] Pflüger's Archiv. VI. 266.

[5] Ibid., IV. 1.

[6] Tom. III. p. 167.

[7] Compare W. Brinton, l. c., and the Lancet, 1859.

The constipation is generally complete after the occlusion has occurred. In some cases spontaneous or induced evacuation of the contents of the portion below the obstruction takes place, and the amount naturally is greater when the seat of the occlusion is higher. In the latter case, diarrhœal evacuations are occasionally observed. When the active peristaltic movements pass beyond the occluded point to the sensitive end of the rectum and excite spasmodic contractions of the sphincter ani, painful tenesmus is produced, which is the more intense the nearer the obstruction lies to the anus, and is notably severe only when the colon is constricted or occluded.

Under the name "cholera herniaire" Malgaigne described cases of strangulated external hernia, in which, together with abundant vomiting, collapse, and the other symptoms of strangulation, profuse diarrhœa occurred. Such cases (occurring also, though very rarely, with internal strangulation) can be explained most simply, I think, by the hypothesis that, as in acute invagination, the permeability of the intestine was not entirely destroyed. (Incomplete strangulation.) Still the possibility of copious diarrhœa with total occlusion is not to be denied, when the latter occurs high up in the small intestine, in the jejunum, or duodenum. If in such a case the active peristaltic movements extend to the portion lying below the occluded point, they promptly force its liquid contents out, and thus produce diarrhœa.[1]

Others, Brinton for example, relying upon cases of occlusion of the intestines in which profuse watery discharges through the anus accompanied free vomiting,[2] believed that in such cases, as in cholera, transudation takes place into the canal. They were disposed to attribute this to paralysis of the abdominal sympathetic and especially of the splanchnic coming on with the shock of the strangulation, which would cause intense hyperæmia of the intestines. But Ludwig's and Heffter's experiments prove that diarrhœa does not immediately follow division of these nerves; it only occurs when irritation of the intestine or of the vagus is added to division of the splanchnic. Furthermore, the diarrhœas produced in this way are only the consequence of increased peristaltic action, and should not be referred to transudation into the intestines. In a small dog in which I succeeded, by following Asp's method, in cutting both splanchnic nerves without injury to the diaphragm, I saw no diarrhœa, nor could I discover, after killing the animal, any increase of the amount of liquid inside the intestine.

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[1] Houckgeest (l. c. 302), after occlusion of the highest part of the small intestine of a rabbit, saw active peristaltic movements in the portion below causing free evacuation of pulpy matter. I have twice repeated this experiment upon rabbits with the same result; two subsequent experiments upon rabbits, and one upon a dog, gave negative results.

[2] This is not unfrequently observed in cases of invagination. Thus one of Herrmann's cases was supposed to be cholera, on account of the profuse passages and the other symptoms, until the vomiting became fæcal. Petersburg. Med. Ztg. 1867. Bd. XII. S. 233.

The adherents of the transudation theory have further appealed, in favor of their views, to the fact that the vomiting which accompanies the profuse diarrhœa is often so abundant that it bears no relation to the amount of liquid taken in by the mouth and estimated to be present in the intestine,[1] and, further, that, sometimes in cases of acute internal strangulation, symptoms pointing to rapid withdrawal of water from the blood, such as dryness of the mucous membrane, intense thirst, choleraic face and voice, cramps in the calves of the legs, and anuria, have been observed almost simultaneously with the first appearance of the disease. But these incidents are most simply explained by the theory that the absorption of water by the blood is notably diminished by the occurrence of internal strangulation, while the loss of water through the skin and lungs, or even by sweating, is increased. The determination of these questions must, however, be reserved for future and more exact observations.

The grave symptoms of collapse accompanying acute strangulation of the intestines correspond in many respects to those which mark the occurrence of shock after a blow upon the abdomen with a blunt weapon, or after perforation of an ulcer of the stomach or intestine. We may call them the shock of strangulation. They can be approximately explained as follows, according to the knowledge furnished by the investigations of Bezold,[2] Goltz,[3] Bernstein,[4] and Ludwig and Asp,[5] of the physiological relations of the nerves of the intestines to the innervation of the heart and vessels.

The injuries caused by the strangulation lead to paralysis of the splanchnic nerve, to reduction of the tonicity of the vessels of the intestine, and to congestion of the abdominal viscera. As a result of this, the rest of the arterial system becomes relatively anæmic, and we have anæmia of the skin, smallness and thinness of the pulse. The vessels of the brain share in the anæmia; and this, as is always the case in cerebral anæmia, leads to diminution of the tonicity of the pneumogastric and consequent quickening of the pulse. Thus in strangulation of the intestine we usually find the pulse not only small, but also, as during a swoon, quicker. The same symptoms follow division of the splanchnic. Occasionally the pulse has been observed to be notably slower immediately after the occurrence of strangulation of the intestine; the same has been sometimes seen during traumatic shock, and I saw it in a case of perforation of the intestine.[1] A violent irritation of the sensory branches of the splanchnic, as well as of the abdominal sympathetic, causes, as Ludwig and Asp have shown, increase of the blood-pressure, increase of the tonicity of the pneumogastric, and in this way slowing of the pulse. But as the slowing of the pulse, occasionally observed during the shock following a traumatism or strangulation of the intestine, is usually accompanied by all the signs of diminished blood-pressure, it should rather be regarded as due to a reflex irritation of the pneumogastric centre proceeding from an irritation (connected with the injuries caused by the strangulation) of the abdominal sympathetic and splanchnic, a reflex process which, it is true, has only been demonstrated with certainty upon frogs, but of which Ludwig and Asp found at least indications in their experiments upon dogs (l. c. S. 174). In any case, the period of slowing of the pulse with reduction of pressure (irritation of the sensory fibres of the splanchnic, reflex irritation of the pneumogastric centre) is followed immediately by quickening of the pulse with reduction of pressure, this being due to paralysis of the vaso-motor fibres of the splanchnic which leads to hyperæmia of the intestines, anæmia of the rest of the body and of the brain, and thereby to reduction of the tonicity of the pneumogastric.

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[1] Compare Brinton, l. c.

[2] Innervation d. Herzens. Würzb. 1863; and Untersuch. a. d. Würzb. Laborat. 1867.

[3] Virch. Arch. Bd. 27 and 28.

[4] Centralblatt. 1864. No. 52.

[5] Beobacht. über Gefässnerv. A. d. phys. Inst. z. Leipzic. Bericht d. math.-phys. Cl. d. k. sächs. Ges. d. Wissensch. 1867. S. 135.

As to the temperature of the body during the different stages of strangulation of the intestines, we posses but scanty data. It is sufficient to touch the body to recognize that the temperature of the peripheral parts is lowered in consequence of the anæmia and the retardation of the circulation. If, now, the blood collects in the abdominal vessels in consequence of the paralysis of the splanchnic and returns more slowly to the surface, and if the production of heat goes on normally, we should expect to find the temperature of the internal parts raised. But as the records of rectal temperature taken during the shock of strangulation generally show a not inconsiderable reduction of the internal temperature, we must infer that the production of heat is diminished, and that this is due to the reflex influence of certain nervous apparatuses. On the appearance of diffuse peritonitis, the temperature of the body rises.

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[1] A few days ago I saw an unusually severe case of obstruction by gall-stones; the patient was covered with cold sweat, had cool extremities, muffled voice, choleraic countenance, vomited freely, and presented a board-like tension of the abdomen. The temperature in the rectum, taken by myself, was 95½°, a thermometer placed at the same time in the axilla and compared with the one in the rectum marked only 92°, the pulse was small, its frequency 48 (!) in the minute. After an injection of morphine, the tension of the abdomen diminished, the skin filled with blood, the pulse rose gradually to 76, the temperature in the rectum, after the patient had passed an hour of comparative comfort, was 99.68°.

The change of the features to the form of the facies hippocratica, the sinking of the eyes, the diminished turgescence of the skin, the aphonia, and the dryness of the skin, are due in part to the diminished absorption of water in consequence of the violent vomiting and to the withdrawal of water from the blood and tissues by the profuse sweats which usually occur simultaneously, and in part to the ischæmia of the skin. The profuse cold sweats can hardly be explained by altered circulatory conditions, and should rather be attributed to paralysis of a nerve-centre for preventing perspiration, an explanation similar to that offered by Immermann[1] for the death sweat and the hyperidrosis of fainting.

The proposition has been advanced, very dogmatically, that occlusions and constrictions which are situated high up in the small intestine are accompanied by anuria or by notable oliguria, and that, on the other hand, occlusions of the lower part of the ileum, and especially of the colon, are associated with the voiding of the normal amount of urine.[2] Laid down in this way, the proposition needs certain limitations. In most cases of acute occlusion of the intestine with serious collapse, oliguria or anuria occurs, no matter where the seat of the obstruction may be, whether in the duodenum, or even in the sigmoid flexure, just as it does in the shock following the infliction of a wound upon the abdomen, or during a violent paroxysm of hepatic or renal colic.[3] The cause of the anuria under these circumstances lies partly in the violent vomiting and in the withdrawal of water from the blood by the free sweating, and partly in the reduced pressure of the blood, due to the shock, in the aortic system, in the glomeruli of the kidneys. And only as occlusions situated high up in the small intestines are accompanied more frequently by symptoms of shock, which are at the same time specially severe, as the vomiting sets in earlier and is more profuse, and, finally, as the absorbing surface of the intestine is considerably diminished, so, too, does total anuria, or a marked degree of oliguria, occur earlier, and on the average, more frequently. The symptom of oliguria has a significance in the differential diagnosis of the seat of the obstruction only in cases of chronic incomplete occlusions or constrictions of the intestines. If these are situated high up, the amount of urine, for the same well-known reasons as in stenosis of the pylorus, is constantly diminished, and this is not the case when the occlusion is in the lower part of the ileum or in the colon.

The mental functions in patients with internal strangulation usually remain quite undisturbed, up to the very hour of death. In some cases, the sensorium becomes affected, and this may go on to sopor and coma. Patients have been occasionally attacked at an early period with violent delirium and convulsions, and exceptionally have even died with trismus and tetanus.[1] The difference in behavior of the mental functions depends upon the slower or more rapid occurrence, and upon the degree of the cerebral anæmia[2] and cerebral œdema. The insufficiency of the respiration, due to the permanent elevation of the diaphragm, and which leads to an excess of carbonic acid in the blood, is often of decisive influence upon the condition of the sensorium. In cases of acute strangulation with anuria, lasting for several days, some have sought to explain the severe cerebral symptoms (coma, convulsions) by blood-poisoning, just as death following coma and epileptiform convulsions, in the last stage of cholera, has been charged to uræmic poisoning.

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[1] Deutsches Archiv f. klin. Med. XII. Bd. S. 179.

[2] For details see *Barlow*, Guy's Hosp. Reports, Vol. II. Series II.; and *Sedgwick*, Med. Chir. Transac. Vol. 51.

[3] Thus in the severe case of hepatic colic mentioned above, only 370 cubic centimetres of dark, red urine (of the sp. gr. of 1,031) were passed during 24 hours.

Symptoms of greater diagnostic value are furnished by *examination of the abdomen.*

As the gases and other contents collect above the occlusion, distention of the corresponding loops and of the abdomen occurs, meteorism. Abnormal gaseous products of decomposition formed by the stagnating contents of the intestine may also aid in its production. The meteorism is the more considerable and general, as the occlusion lies deeper in the intestine. It is augmented by the occurrence of peritonitis and paralysis of intestinal muscles. Not only does the distention of the abdomen vary in intensity according to the seat of the obstruction, but it is also limited to certain regions, a relation which offers important aid in making the diagnosis of the seat of the occlusion, but which also can lead into error.

If the lower part of the colon or the rectum is occluded, the distention is often limited at first to the region occupied by the colon, the loins, hypochondria, and epigastrium, and the transverse colon especially projects like a well-defined tumor, while the meso- and hypogastrium are relatively sunken. But in such cases, as we have already said, the ileo-cæcal valve soon becomes insufficient, and the meteorism becomes general and regular. Even when the meteorism is strictly limited to the colon, the distention of the abdomen may be general. This happens when, as is frequently the case in chronic constrictions of the lower colon, its upper part is thrown into numerous convolutions, occupying not only its usual place but also the whole meso- and hypogastrium, and completely covering the small intestine. Indeed exceptional cases have been seen of meteorism of the colon alone, in which the hypo- and mesogastrium were more distended than the region of the transverse colon was, and therefore it was thought to be meteorism of the small intestine alone. This is the case when the transverse colon is drawn downwards to the symphysis, so as to form with the ascending and descending colons the letter M.

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[1] For example, in a case of twisting of the sigmoid flexure (Petersburg Med. Ztg. N. F. I. 1870. S. 565); also in another case of twisting (Bericht a. d. k. k. allg. Krankenh. in Wien. 1858. S. 331). In cases of intussusception in children, violent convulsions are often observed at the beginning as well as at the end of the disease.

[2] Compare *Niemeyer*, Lehrb. d. spec. Path. u. Therap. 7. Aufl. 2. Bd. 1868.

When the occlusion is seated in the lower part of the ileum or in the cæcum, the distention is limited to the small intestine, and the meso- and hypogastrium are usually more swollen than the region of the colon square. But considerable tympanites of the convolutions of the small intestine forces the colon backwards, and extends with general distention of the abdomen into the regions corresponding to the colon. There is one sign which is always absent, but which, on the other hand, cannot always be made out in meteorism of the colon—that is, the visible projection of a swelling running across the epigastrium and corresponding to the transverse colon. When the obstruction is seated high up in the jejunum or the duodenum, the meteorism is entirely absent, or is limited to the epigastrium, while the rest of the abdomen seems sunken. When the obstruction remains for some time in the highest portions of the small intestine, the consecutive ectasia of the stomach, as I had occasion to again observe a few days ago, may increase to such an extent, that meteorism of the meso-, and even of the hypogastrium results. The higher the seat of the obstruction, the more do we often see a sudden change in the degree of meteorism after a fit of vomiting, a fact which deserves more attention than has heretofore been given to it. I have yet to mention irregular asymmetrical meteorism, in which one side of the abdomen usually is more swollen than the other. This greater distention occupies the right side when the obstruction is seated between the hepatic bend and the upper part of the descending colon, and the left side in cases of stenosis of the rectum, and especially of ileo-cæcal or colon intussusceptions which extend into the colon or rectum.

A point of great diagnostic value is the distinctness of the outlines of the convolutions and their peristaltic movements, as seen through the abdominal walls. We shall discuss this symptom among those of constriction of the intestine, because it appears only exceptionally in acute internal incarcerations, and, on the other hand, very frequently in chronic constrictions, and in the occlusions which result from them.

Other symptoms are furnished by palpation, percussion, and auscultation of the abdomen. *Palpation* enables us but very rarely to detect a tumor or an increase of resistance in cases of internal incarceration. On the other hand, this is almost always the case in occlusions by intussusception, by compressing abdominal tumors, neoplasms, and masses of fæces. Careful examination by palpation of the different hernial orifices furnishes important help in distinguishing between internal incarceration and strangulation of an external hernia. The digital, or *manual examination of the rectum* and vagina, furnishes, under certain circumstances, symptoms of great diagnostic and therapeutical value, and the old rule, to make a careful examination by palpation of the hernial orifices and rectum, and then of the vagina in all cases of occlusion of the intestine, cannot be too often repeated. We shall refer to this again when speaking of the symptoms of constriction of the intestine.

Percussion of the abdomen is of subordinate importance in making the diagnosis of occlusion of the intestine, or of its situation. When the meteorism is marked, the percussion note is of the same intensity and same character over the entire region occupied by the distended bowels—that is, it is sonorous, "non-tympanitic," or "deep-tympanitic," drum-like. When the meteorism is more localized, the parts over-distended by air can be distinguished by their sonorous, non-tympanitic note from the less distended parts, which give a higher, clear ringing, often metallic-like percussion note. And also when, by the usual method of percussion, the whole abdomen gives a note of the same intensity and same characters, percussion upon a pleximeter, with simultaneous auscultation, often brings out differences in tone, which make it possible to distinguish different parts of the intestines from each other. In cases of chronic constriction, notwithstanding a notable distention of the abdomen and intestines, the percussion note is often a ringing one everywhere. This is the case when the abnormal filling with gas is associated with only a slight tension of the already over-stretched intestinal and abdominal wall. The percussion note of course is dull at points where non-gaseous contents are collected, especially, therefore, in the dependent, lateral portions of the abdomen. If this dulness is due to liquid, easily movable contents, it will sometimes give place to a clear percussion note when the patient's position is changed, and thus the real condition may be mistaken for an effusion of liquid into the cavity of the abdomen. As the meteorism increases, the liver is forced further and further upwards, and rotated backwards about its transverse axis, so that finally it touches the anterior wall of the body nowhere, and the hepatic dulness is no longer to be made out. The elevation of the diaphragm, which often causes considerable dyspnœa and cyanosis, with purely supra-costal respiration, is further shown by the fact that the posterior and lower borders of the lungs, as can be easily shown, especially upon the right side, are higher than usual, and that not unfrequently the apex-beat of the heart also is to be felt in the intercostal space above the usual one.

The following phenomenon on percussion is found not only in occlusions and constrictions of the intestines, but also not unfrequently in simple diarrhœa, after the administration of purgatives, and, under normal conditions, a few hours after having taken food: if percussion is made for a long time on one and the same point of the abdomen the ringing percussion changes, runs up and down the scale, and varies from thin to full and back again, or it changes suddenly from a high note to a low one, from a clear one to a muffled one. While the first is due to gradual change in the distention and amount of gas in the one loop percussed, the other is due to the sudden substitution of one loop for another, under different conditions of distention. Under certain circumstances this may furnish an approximative measure of the vigor of the peristaltic action.

As for *auscultation* of the abdomen, we have to mention only those loud borborygmi which can be heard by the patient, and also by those standing at some distance from him, and which are produced by the active peristaltic movements of the intestines filled with gas and liquid. When the sounds are made by the gas alone, they are dry and cooing; when gas and liquid are mingled, they are moist, splashing, and like the manifold sounds produced by pouring liquid into a bottle, or by shaking half-filled bottles. Usually they are first heard by laying the ear against the abdomen; then they become astonishingly loud and abundant, often lasting continuously for hours, and give an idea of the vigor of the peristaltic action. After the administration of opium, or the injection of morphine, they become quieter, or—as I have repeatedly noticed in patients with diarrhœa—fewer, and with longer intervals. The same sounds can often be produced by shaking the patient, or changing his position, or by rapid bimanual palpation, or by quick, vigorous percussion. The hand laid flat upon the abdomen sometimes feels the movements of the liquid contents and the trembling of the tense intestinal walls.

Diagnosis.

The diagnosis of occlusion of the intestines follows from the train of symptoms just described.

We have first to consider the diseases with reference to their differential diagnosis. Since we are now occupied only with internal incarcerations, we must consider the means of distinguishing them from strangulated external hernia. It is a rule as old as it is important, and now seldom omitted, on account of the stress that has been repeatedly laid upon it, *always to make a careful examination of the different points at which hernia may occur in every patient who presents symptoms of acute strangulation of the intestines.* The physician will never allow himself to be restrained from making such an examination by the patient's assurance that "he has never had a rupture." The discovery of any of the usual external herniæ ordinarily presents no difficulty; but it is not so with the diagnosis of a hernia foraminis ovalis, of a small interstitial or intra-parietal inguinal or femoral hernia, or of the very rare hernia lumbalis, ischiatica, etc. The latter are very difficult to recognize, and indeed often pass for "internal strangulation," their real character being first discovered during the autopsy.

When we call to mind the symptoms of acute incarceration in its severest form, the sudden attack with cholera-like collapse, thready pulse, cyanotic cool extremities, choleraic countenance and voice, violent vomiting, anxiety, and suppression of urine, when we add to that its sometimes rapidly fatal course, we understand how even good observers in cholera seasons may have mistaken cases of internal strangulation for cholera, and only found out their error at the autopsy.[1]

But still mistakes of this kind are extremely rare, and indeed are only possible when the course is very acute and the occlusion seated high up in the jejunum or duodenum; for only in such a case is the meteorism lacking, the vomiting copious and slightly colored, the collapse rapid, and suppression of urine frequent. But even then the obstinate constipation and the violent colicky pains protect against error.

Cases are repeatedly mentioned in which acute internal incarcerations excited suspicions of poisoning, especially with arsenic, to be relieved only by the official examination of the body.[2]

It is much less difficult to distinguish acute incarceration from cholera or poisoning than it is from other abdominal affections which come on with severe symptoms of collapse, violent pains, meteorism, vomiting, and constipation. We have first to mention the marked resemblance offered by an unusually severe acute typhlitis, and then that of a péritonite foudroyante, such as is caused by perforation of the appendix,[3] of an ulcer of the stomach or intestine,[4] or by many other causes.

The symptoms of an occlusion of the intestine situated high up, in the duodenum, for example,—acute collapse, violent non-feculent vomiting, the abdomen retracted rather than distended —can be closely imitated by a violent attack of hepatic or renal colic. In a case of acute occlusion of the jejunum reported by Rees,[1] meningitis was suspected on account of the delirium, the violent vomiting, and the retracted abdomen. The mistake is still more easily made in children, for in them a simple colica flatulenta or verminosa may give rise to the severest symptoms with collapse, convulsions, vomiting, meteorism, etc. In adults also we sometimes see an unusually severe "colic," with all the symptoms of internal incarceration, except stercoraceous vomiting, terminate favorably. Many of these colics depend upon temporary occlusions of the intestine, and very many of the "recoveries from ileus" so often mentioned in medical literature are nothing but unusually severe temporary obstructions by fæces or other intestinal contents, with hyperextension of the intestines lying above, and peritoneal irritation excited thereby. I remember a case, that occurred in my early practice, where the drinking of an uncommon amount of new, badly fermented beer caused the severest symptoms, exactly like those of internal strangulation. Finally, I need not remind you that the severe symptoms of shock, such as sometimes follow an injury of the abdomen, a contusion of the testicle, etc., are remarkably like those of acute incarceration. The history of the case and its subsequent course prevent mistakes.

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[1] For example, *Fournier* and *Ollivier*, Gaz. Méd. de Paris. 1868. Such a mistake is still more easily made if the strangulation is accompanied by diarrhœa. See "Cholera Herniaire," page 497. Compare, also, the interesting hyper-acute case in the Aerztl. Ber. d. k. k. allg. Krankenh. zu Wien. 1866. S. 145.

[2] *Tardieu*, Annal. d'Hyg. July, 1854. *Gualth van Döveren*, Spec. obs. acad. Groning et Lugd. Bat. 1765. *Basedow*, Casp. Wochenschr. 1837. N. 20.

[3] Arch gén. Oct. 1874. p. 458.

[4] For example, *Lingen*, Petersb. Med. Zeitschr. 1870. I. S. 169.

The diagnosis in cases of internal strangulation aims, however, not only to distinguish it from other affections presenting similar symptoms, but it seeks also to determine the *seat* and the *cause of the occlusion*. Excepting those cases in which the seat of the occlusion can be directly reached by manual or instrumental examination of the rectum, or in which this can be determined from the position and shape of a tumor that can be felt through the vagina or the abdominal walls (*e. g.*, invaginations or fæcal tumors in the course of the colon), we find in internal incarceration the following signs, by the aid of which we can form, under certain circumstances, an opinion as to the seat of the obstruction :

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[1] Fagge, l. c. S. 323.

1. In occlusions of the upper part of the small intestine (jejunum or duodenum) the symptoms of collapse, vomiting, and anuria usually appear early, and soon reach a considerable height. The course is usually rapid, the meteorism inconsiderable and limited to the epigastrium, or it is entirely lacking, and the abdomen may even be retracted; sometimes (if constriction has existed for some time previously) we find the signs of ectasia of the stomach. The vomited matter is constantly stained with bile, greatly discolored, never feculent. The meteorism of the epigastrium is subject to great changes, and especially becomes much less immediately after the vomiting.

2. When there is occlusion of the lower part of the ileum, the meteorism is noteworthy, sometimes limited mainly to the meso- and hypogastrium, with comparative hollowness of the regions corresponding to the colon. Here, too, the course is usually rapid; collapse, vomiting, suppression of urine, appear early. The vomited matter soon becomes feculent.

3. The course of occlusions situated in the lower colon is often less rapid, the vomiting, the collapse appear later, the vomited matter is always feculent. The meteorism is great. At the very beginning it is often limited to a roller-like prominence along the line of the colon; afterwards it spreads over the entire ileum, and reaches the highest degree of general meteorism.

In addition to this, we have the often definitive results of examination of the rectum (by the hand, sounds, and injections) and of the vagina. But it would be a great mistake to suppose, as is so often done, that occlusions of the colon always present a slower course, with tardy collapse, late vomiting, and absence of anuria. This is true only of the more frequent kinds of occlusion of the rectum and colon, such as those produced in consequence of obstruction by fæces (more rarely also by foreign bodies, enteroliths), and those which have been superadded to old constrictions of various kinds or to chronic compression by a tumor. On the other hand, experience shows that twisting of the colon, its acute compression, or its rare strangulation by false ligaments, etc., runs a course as rapid and with as violent vomiting, collapse, and anuria, as that of incarceration of the ileum, and hence we see very plainly that the severity of the attack, the intensity of the collapse, the time of its appearance, etc., are dependent not only upon the seat of the obstruction, whether this is higher or lower in the intestine, but also upon the degree of mechanical injury inflicted upon the peritoneum of the intestine and its mesentery by the cause of the occlusion.

As for the diagnosis of the cause of the occlusion, we shall speak of that with more detail when considering the different anatomico-pathological processes which lead to occlusion. In a number of cases a definite diagnosis is possible. This is true of those which can be reached through the rectum, those due to compression by tumors which can be felt through the vagina or the abdominal walls, and whose existence may have been previously known to the physician and patient. Other causes are sometimes revealed by characteristic signs; this is the case in intussusceptions, large diaphragmatic hernias. The causal diagnosis can also be made in those occlusions which follow old chronic constrictions, the nature of which has been diagnosed (dysentery, neoplasms, separated intussuscepta, tuberculosis, etc.) Sometimes the history of the case supplies definitive information, as when foreign bodies have been swallowed or pushed up into the rectum; at others it furnishes important facts, which are valuable at least for the formation of a probable diagnosis, as when it points to an earlier abdominal or pelvic peritonitis, a typhlitis, an external hernia that has been strangulated and then reduced, previous hepatic colics, perhaps with subsequent evacuation of gall-stones, previous passage of intestinal stones (Monro). But distinctive anamnestic information, or characteristic objective signs, are often lacking, and our diagnostic ability is limited to this, that, by a consideration of the history, of the course of the disease, of the seat of the occlusion, of the age and sex of the patient, and of the relative frequency of the different causes of occlusion, we can reduce the circle of possibilities by exclusion, and form a more or less well-grounded hypothesis, while diagnosis, in the exact sense of the word, does not advance beyond a determination of the existing occlusion and its probable position.

Terminations, Duration, and Prognosis.

There is no cause of acute occlusion of the intestine which cannot spontaneously disappear as well as originate. An intestinal knot can loose itself, an incarcerated or strangulated loop can become free, an invagination can become disengaged, compression cease, twisting or dislocation of the intestine with angular bend can straighten itself, a lodged gall or intestinal stone or foreign body may be dislodged and evacuated, and severe fæcal obturation may be overcome. The not infrequent examples—two such have happened in my own experience—of individuals restored to health after presenting all the symptoms of internal strangulation, including stercoraceous vomiting, give but seldom (*e.g.*, in cases of evacuation of gall-stones or enteroliths, in intussusceptions, etc.) any information as to the cause of the occlusion. But the number of these spontaneous cures is exceedingly small when we compare them with the mass of cases that have ended fatally, and we must describe the prognosis of internal occlusion of the intestine as in the highest degree unfavorable, and ileus as—to use De Haën's words—a "morbus terribilis, creberrime letalis." As for other points of prognosis, excepting in occlusions by gall-stones, enteroliths, foreign bodies, and intussusceptions, all other causes (*e. g.*, false ligaments, diverticuli) are of such a kind that they remain even after a restoration of the permeability of the bowels, and, according to our experience, rather increase than diminish the danger of a relapse. But even with the restoration of the permeability of the intestine, the recovery of the patient is not yet assured. The partial peritonitis remaining at the seat of the occlusion may become general, or it may reduce the calibre of the intestine by constriction or adhesions, and thus give rise to new dangers, and not unfrequently, after a longer or shorter time, to total occlusion or to death in some other way. This latter sometimes follows immediately after freeing of the strangulated intestine, because the loops, being too much injured to be able to recover, go on to gangrene and perforation. A cure, which usually is but temporary, and in extreme cases incomplete, is effected by the formation of a fistula bimucosa between portions of the intestines above and below the occlusion, or by an external opening with formation of an anus prænaturalis (abdominalis or vaginalis).

The most common termination of occlusion of the intestine is by fatal general peritonitis with or without perforation.

The appearance of diffuse peritonitis is indicated by the increasing distention of the abdomen with projection of the borders of the ribs and hypochondria, by the augmented dyspnœa, disappearance of the region of dulness over the liver, the more frequent vomiting, singultus, intensified collapse, rise of temperature, frequency and smallness of the pulse, exacerbation of the pains, and the appearance of diffuse sensibility to pressure on the part of the abdomen. With the onset of diffuse peritonitis, the patient, who hitherto has been in a condition of constant jactitation and restlessness, usually lies quietly upon his back. After the symptoms of peritonitis have lasted for some time, we often find evidence of liquid exudation in the dependent parts of the abdomen.

In the same way, but much more suddenly, is the train of symptoms affected by the occurrence of perforation. Occasionally this is introduced by severe symptoms of shock, sometimes by a profound fainting-fit. The meteorism becomes intense in a very short time. If the liver has not already been separated from the anterior abdominal wall by the meteorismus intestinalis, it becomes so immediately upon the occurrence of the perforation. Partial or unsymmetrical distention of the abdomen, or the outline of the convolutions showing through the walls, disappears; the abdomen becomes equally prominent everywhere. The evidence of freely-movable liquid within the peritoneal cavity, which before was lacking, is now to be had. Sometimes the pain ceases as soon as the perforation takes place, probably because the partially relieved intestines, whose increased peristaltic action excited pain, are now at rest. In other cases, when there has been no previous general peritonitis, the perforation excites intense pain; while in patients with unaffected peritoneum perforation often excites at once the most intense collapse, with loss of consciousness, lasting until death; in other cases comatose patients are aroused by it from their coma.

Among the final phenomena are sometimes the pneumonias due to foreign bodies, those caused by the inspiration of vomited matter. In consequence of the inflammation, fæcal infiltration, and mortification of the retro-peritoneal cellular tissue, or when there is extensive ulcerative destruction of the intestines, the absorption by the blood of putrid elements from the decomposed contents of the intestine sometimes gives rise to symptoms resembling septicæmia, or embolic pulmonary infarctions follow from the decomposition of the retro-peritoneal cellular tissue. In consequence of these processes, irregular chills, high fever, and pulmonary symptoms (such as those of pleurisy or infiltration of the lungs, stabbing pain in the chest, cough with bloody expectoration) appear as the end approaches.

In exceedingly rare cases embolic abscesses of the liver have been found under the above conditions (Coze, Reid). Now and again mention is also made of icterus by obstruction (Fine, Warren), which is explained by the pressure of the distended intestines upon the concavity of the liver and the bile ducts leading from it (?).[1]

Death in cases of occlusion of the intestines—whether general peritonitis or perforation precedes or not—is in most cases due to arterial anæmia of the brain (often associated with cerebral œdema), and to consecutive paralysis of the heart and respiration. Sometimes death occurs suddenly—for example, while rising in bed (in consequence of fatal increase of the cerebral anæmia), or after severe fainting fits have repeatedly followed similar occasions.

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[1] Compare the following account of the symptoms of Coprostasis.

The duration of acute occlusion of the intestines, in general until death, is on the average six days,[1] and varies from eight hours[2] to thirteen days. It depends upon the kind of anatomical process which produces the occlusion, and upon the course, especially upon the time of the occurrence of peritonitis, gangrene, and perforation. The patient's age, constitution, and power of resistance, as well as the mode of treatment, must also be taken into consideration. The higher the seat of the occlusion, the more acute, generally, is the course. But, as we shall see, there are also some occlusions of the colon, such as acute twisting of the sigmoid flexure, which, as regards the acuteness of their course, are not inferior to occlusions situated high up, and we find in this a confirmation of what was previously stated, that the duration, like the course, is determined, not alone by the situation of the occlusion, but also by the degree of the mechanical injury inflicted at the occluded point.

Pathology of Constrictions of the Intestines.

Symptoms.

We include in this not only stenoses of the intestine in the narrower sense of the word, but all those various causes which partly interfere with the permeability of the canal and impede the natural advance of the contents of the bowels.

In contra-distinction from internal strangulations, which have usually an acute origin and an acute course, we have here to deal with diseases which are usually developed gradually and insidiously, and have a chronic course. This is especially true of strictures in the narrower sense of the word. On the other hand, it sometimes happens that internal incarcerations cause total impermeability at first, and appear with the severe symptoms of acute strangulation of the intestine, but then pass into a chronic state of only partial occlusion or constriction. Under such circumstances we speak of chronic incarceration[1] by false ligaments, hernial openings, etc., of compressions of acute origin but chronic course, of partial obstruction by gall-stones, intestinal stones, foreign bodies, and intussusception. Of intestinal knots alone do I know of no chronic case of this kind.

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[1] Average of three hundred and fifty cases of internal strangulation.

[2] *E. g.*, *Delaye*, L'Union méd. 1855. p. 467. Aerzt. Bericht aus d. k. k. allg. Krankh. zu Wien. 1862. S. 78.

On the other hand, it not unfrequently happens that a constriction of the intestine remains entirely latent for a long time and then suddenly, upon the accession of certain conditions, of which we shall hereafter speak in detail, goes on to sudden occlusion, with all the symptoms of internal incarceration.

We see from the above that the manner varies in which alterations of the permeability of the intestines appear, and that the rule, frequently laid down as being without exception, that the causes of constriction always begin chronically and gradually, those of incarceration, on the other hand, suddenly, offers frequent exceptions.

The symptoms of constriction of the intestines vary according to the seat, the anatomical cause, and the degree of the constriction.

The symptoms of stenosis, ceteris paribus, are more marked and appear earlier, as the contents of the intestine, which have to be moved through it, are of firmer consistency. Stenoses in the colon and rectum, therefore, often manifest themselves early and with disproportionately severer symptoms than constrictions of the same degree in the ileum, where the thin, liquid contents pass through them. Constrictions in the ileum not unfrequently remain latent for a long time, and only manifest themselves when they have reached a high degree; but then their course is so much the more acute. Constrictions in the colon or rectum manifest themselves promptly, even when the narrowing is still slight; but their course is so much the more chronic.

It has certainly happened to many observers to find at an autopsy a not inconsiderable constriction at some point of the small intestine, which had presented no symptoms during life. I can recall two cases of this kind that have happened during the last few years. In one case a carcinoma of the uterus, in the other a dermoid cyst of the ovary had become adherent to the ileum and had diminished the calibre of the intestine, in the first case to the size of the little finger, without giving rise to any symptom whatever on the part of the intestine during life.

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[1] *J. Wagner*, Med. Jahrb. d. österr. St. 1833. Bd. IV. *Fagge*, l. c., S. 349.

Stenosis causes the more symptoms the more suddenly it is produced, for then compensation for the obstruction, by hypertrophy of the muscular coat of the intestine above it, has not made the same progress.

Every constriction retards or obstructs the passage of the contents of the intestines. The arrest of the gaseous, liquid, or solid contents leads to increased distention of the intestine above the obstruction and of the abdomen—meteorism. This is the more considerable as the passage through the constriction takes place more incompletely, the arrest lasts longer, and the obstacle is situated lower. Meteorism is lacking, the abdomen is sunken, and the stomach sometimes dilated when the stenosis is high up (in the jejunum or duodenum). Under such circumstances the differential diagnosis from stenosis of the pylorus is not unfrequently impossible, and the more so because in duodenal and jejunal stenoses vomiting occurs almost as frequently, and the secretion of urine is almost as constantly diminished as in stenosis of the pylorus.

Perhaps the continuous presence of large amounts of bile in the vomited matter might enable us to distinguish between stenosis above and below the orifice of the ductus choledochus. In the year 1869, when assistant at the Lindwurm Clinic, I saw a woman between fifty and sixty years old, with retracted abdomen, considerable gastric ectasia, visible peristaltic movements of the stomach, abundant, constantly dark-green vomiting, with an immovable, deeply-seated tumor near the umbilicus, great emaciation, cachectic color of the skin, obstinate constipation, and constant oliguria. Death followed, under conditions of the most extreme desiccation, after several days of anuria, aphonia, great dryness of the tongue, cramps in the legs, etc. The diagnosis inclined towards cancer of the stomach. The autopsy showed carcinoma of the retro-peritoneal glands, which had drawn upon the flexura duodeno-jejunalis, and narrowed it to the size of a lead-pencil.

As for the dependence of the form and degree of the meteorism upon the seat of the stenosis, we refer to the detailed discussion of the same question under Occlusion of the Intestines. Meteorism in intestinal constrictions varies in degree extraordinarily within short periods of time; after free evacuations it is inconsiderable, and again very marked after arrest has lasted for some time. Meteorism is associated with a series of subjective annoyances, with the sensation of fulness in the abdomen, pressure in the hypochondria, fetid eructations, shortness of breath, palpitation of the heart, and pains in the back radiating towards other regions. The increased peristaltic pressure towards the obstruction, united with the over-distention and tenseness of the intestinal walls, occasions frequent paroxysms of colic, especially after stoppage of the contents has lasted for a long time.

A frequent and diagnostically important symptom of long-standing stenosis is the conspicuousness of the outlines of the convolutions and their peristaltic changes in size and situation, as seen through the abdominal walls. In cases of chronic constriction this appearance is the more distinct, as the abdominal walls are generally emaciated, and, in consequence of the repeated over-distentions, are lax and not tense, even when the meteorism is very great. Sometimes it is possible to grasp portions of the hypertrophied intestine, when they are in a state of contraction, through the abdominal walls, as if they were stiff tubes.

Under other, partly normal, conditions also the outlines of the convolutions can be faintly seen through the abdominal walls. This is the case when the walls are thin and relaxed, and when, at the same time, the peristaltic changes in the size and situation of the intestine are active. This appearance can be seen in emaciated individuals, especially in women who have just been confined, also in consumptives with diarrhœa, and in feeble children suffering from the same affection. In such cases the flaccid, thin walls lie like a loose cloth over the hills and valleys of the intestinal convolutions; they rise and fall without opposition when the underlying loops sink or contract, swell or are pressed up.

But the phenomenon shows itself much more vigorously in certain cases of constriction where the convolutions can be made out, with exceptional distinctness, even through walls distended by meteorism. This indicates that the resistance of the loops to pressure has increased, and it occurs especially in cases of long-standing obstruction when the intestinal tube has become stiffer and more powerful, not only in consequence of the increased intestinal pressure, but also of the thickening and hypertrophy of its walls.

Hypertrophy of the muscular coat renders active changes in the size and situation of the convolutions possible under conditions of intra-intestinal pressure which would constitute a real obstacle to peristaltic action in an intestine which was not hypertrophied. Hence we see in chronic constrictions and the occlusions which result from them, even high degrees of meteorism, associated with active, puckered, wavy and worm-like, sometimes peculiarly jerking, quivering, or darting movements of the visible convolutions, while in acute occlusions the meteoristic abdomen is distended smoothly and equally. This may sometimes throw important light upon the question whether we have to deal with an internal strangulation that has suddenly arisen, or with an obstruction of long standing and perhaps hitherto latent course.

We can also sometimes draw important conclusions regarding the seat of the occlusion from the outlines of the convolutions as seen through the abdominal walls. Thus, when the transverse colon appears as a distinct transverse swelling, or when the hypo- and mesogastrium are filled with the outlines of many convolutions while the region of the colon is comparatively sunken, we infer, in the first case, that the seat of the obstruction is in the descending colon or rectum, and, in the second, that there is stenosis of the lower part of the ileum or cæcum. But all that was said, when we were speaking of occlusions of the intestines, with reference to these signs, might be repeated here, and especially should it be remembered that in cases of constriction of the lower part of the colon or rectum, the colon often becomes longer and is thrown into unusually free folds, which fill the entire meso- and hypogastrium and show their outlines and peristaltic movements through them. The more numerous and shorter, the more limited and variously directed the swellings showing through the abdominal walls, the more abrupt and vigorous their peristaltic movements, the more abundant and moister the borborygmi heard in them, the more surely can we then conclude that the swellings belong to loops of the ileum.

By auscultation of the abdomen, and often when standing at some distance, we can hear numerous borborygmi, which are produced or increased by succussion, or by changing the position of the patient, or by bimanual palpation, and can often be heard continuously for hours if the ear is laid against the abdomen.

Among the most prominent symptoms of constriction of the intestine are anomalies in the fæcal evacuations. They consist most frequently of constipation, lasting for many days, accompanied by increase of the meteorism and subjective troubles, and followed spontaneously, or after the employment of remedies, by abundant, often extraordinarily fetid diarrhœal evacuations. In other cases, especially in occlusions of the rectum, the constipation alternates with evacuations of hard scybala, often streaked with blood, and accompanied by painful tenesmus. Finally, those cases are far from rare, especially in stenosis of the ileum, but also in those of the colon, in which diarrhœal passages occur several times daily throughout the whole course of the disease, or for a long time; while the meteorism present at the same time, and the abundant moist borborygmi which can be heard by auscultation of the abdomen, prove that the diarrhœal evacuations, notwithstanding their frequency, are insufficient, and that the advance of the contents is still obstructed. The cause of diarrhœa in intestinal stenosis is to be found in the increased peristaltic action, which is indirectly produced by the latter and expels the contents as soon as they have made their way through the constriction, thus preventing their fæcal thickening in the colon. This increased peristaltic action, which extends to the portion of intestine lying below the stenosis (as is shown by the frequent diarrhœa in stenosis of the small intestine), is set up by the irritation of the arrested contents, or by that of ulcers of the mucous membrane at or above the constriction.[1]

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[1] A dogma has been presented and maintained upon this point, also, that when constant diarrhœa is associated with constriction of the intestine, ulcers must be supposed to be the cause. Many, however, have satisfied themselves that this is not necessarily the case, that diarrhœa can occur in stenosis without the existence of ulcers.

In many cases of constriction of the rectum or colon the appearance of the fæces is remarkable. They are composed of flattened, sometimes ribbon-like masses, or of cylinders of the size of a lead-pencil, or present a conglomeration of round or spindle-shaped particles of the size of sheep or goat dung. When the fæces appear in such a form continuously or for a long time, an obstruction in the rectum or colon may be inferred. Still it must be remembered that the same appearance of the fæces is sometimes found when there is no stenosis of the colon or rectum, especially when the colon is tightly contracted in the form of the "starvation intestine,"—that is, under conditions of inanition below constrictions of the ileum or colon, and, as I have repeatedly seen, in cases of stricture of the pylorus. This same appearance is also occasionally observed under conditions associated with violent rectal tenesmus, such as are produced in perfectly healthy individuals by causes that are not well understood.

The admixture of blood or of bloody mucus with the evacuations, or actual hemorrhages, must always excite suspicion of hemorrhoidal origin.[1] They occur especially in certain kinds of intestinal constrictions, most frequently and almost always in intussusception, also in cases of malignant neoplasms, polyps, and especially in villous tumors of the canal. Among the processes of acute internal strangulation only the formation of a knot between the ileum and the sigmoid flexure and the twisting of the latter[2] occasionally lead to bloody evacuations. We must, however, mention that stenosis of the ileum, and even of the upper portions of the colon, may exist with perfectly natural stools.

Most stenoses lead sooner or later—most promptly in the case of malignant neoplasms—to general disturbances of nutrition. The patients grow thin, their complexion becomes pale, sometimes of a dirty yellow, and the features sharper (facies abdominalis). The voice, as is the case in most chronic diseases of the abdomen, is often peevish, irritated, sometimes very hypochondriacal.

Terminations, Duration, and Prognosis.

Every constriction can lead, sooner or later, and also suddenly, the symptoms gradually becoming more severe, to definitive occlusion. Sudden occlusion is caused by the lodging of a foreign body (especially a fruit-seed) or a hard piece of fæces in the constricted part, or by the addition of a twist or kink in the intestine, as is seen especially in strictures of the junction between the rectum and sigmoid flexure, where the kink is caused by the sinking of the heavy S-shaped loop into the lower cavity of the pelvis.[1] Impermeability can also be caused by paralysis of the intestine above the stenosis, resulting from the complication of acute local peritonitis.

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[1] Hemorrhoidal tumors are a frequent secondary symptom, especially in chronic stenosis of the colon.

[2] *Lingen*, Petersb. med. Zeitschr. 1870. Bd. 1. S. 565.

A constriction of the intestine may remain completely latent for a long time, or may present only the slightest symptoms, and then suddenly appear with all the severest indications of internal strangulation. Numerous recorded cases prove this, exceptionally even in stenosis caused by malignant neoplasms.[2] In these cases the diagnosis is generally wrong. The distinctness with which the outlines of the intestines can be seen through the abdominal walls is of great diagnostic value in such cases, for it indicates a long standing though latent diminution of permeability which has led to hypertrophy of the walls of the intestine.

Gradual occlusion is brought about by increase of the stenosis and increasing stoppage of the contents. Since the latter are received more abundantly than they are expelled, the movement of the mass becomes slower and slower; finally complete arrest takes place, because the usual peristaltic pressure is no longer sufficient to force the contents, which, in stenosis of the colon, have become a solid fæcal column, through the obstruction. Such a coprostasis, associated with severe symptoms, is often relieved after symptoms of ileus have already appeared. This is the result either of an unusually strong peristaltic pressure exerted by the hypertrophied intestine, or of the fact that the irritation of the arrested masses has increased the catarrh of the mucous membrane, and produced free secretion of intestinal mucus, which has soaked into the fæces, softened them, and favored their progression. The same process of softening and mobilizing the arrested fæces takes place still more frequently, because the active peristaltic action, excited by the stoppage, forces the liquid contents of the small intestine rapidly into the colon, where they do valuable service in diluting the stagnated contents. In stenosis of the colon the same result can be obtained by injections, which may serve to free the passage when a piece of fæces or a fruit-seed has lodged in and closed the orifice of the stenosis. If the stoppage above the stenosis is relieved in the manner above described, very profuse, offensive diarrhœal discharges follow, often for several days, and thus the constriction of the intestine presents the symptoms of alternating diarrhœa and severe constipation. This condition often lasts for a long time, during which normal evacuations may occasionally occur, but finally it results in permanent occlusion. This results from failure of compensation following degeneration of the hypertrophied muscular coat of the intestine above the stenosis. It is true that the arrested contents now stimulate the intestine to increased efforts, but the latter soon becomes tired, its contents are only partly removed, and an increasing and stagnating residue is retained. The occlusion lasts longer each time, the diarrhœal evacuation comes less frequently, lasts a shorter time, and is more insufficient. The accompanying dilatation of the intestine, which is all the more easily distended because it is degenerated, opposes a new mechanical obstruction to its successful peristaltic contraction. Finally, permanent occlusion results with hyperextension of the paretic intestine. In other cases, sloughing of the mucous membrane and the consecutive, often numerous ulcers above the stenosis, or circumscribed inflammatory processes in the peritoneum itself, play an important part in setting up paralysis of the intestine, for these processes are accompanied by serous imbibition and swelling of the intestinal layers, including the muscular one.

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[1] *Lingen*, Petersb. med. Zeitschr. N. F. 1. Bd. S. 169. *Shaw*, Lond. Path. Soc. IV. Vol. 147; and others.

[2] *Wahl*, Petersb. med. Ztg. 1867. S. 239.

Death in a case of constriction of the intestine can, however, take place without occlusion, in consequence of the occurrence of general peritonitis, with or without perforation. The latter has often occurred at a point far above the obstruction, for example, in the cæcum in stenosis of the rectum, and under such circumstances has its origin in an ulcer following gangrene, caused by pressure of the fæces. Perforation into the retroperitoneal cellular tissue leads to the formation of pouched or tubular decomposing purulent collections, which may make their way to the surface after the manner of migratory abscesses, and give rise to stercoral fistula. We have already described how, under these circumstances, septic blood-poisoning, with high, irregular fever, decomposing venous thromboses, and similar pulmonary infarctions with thoracic symptoms and repeated chills, or the spontaneous formation of an anus præter naturam, a fistula bimucosa of the intestine, or perforation into one of the natural cavities (vagina, uterus, bladder) may take place.

Not unfrequently, especially in stenosis due to malignant new formations, death is caused by increasing apyretic marasmus, by inanition, often after a painful thrombosis in one or both femoral veins, a bedsore, or a fatal pneumonia has appeared.

But a termination by recovery is not impossible in certain kinds of constrictions. Thus, alterations of permeability, which arise from incomplete obstruction, may be permanently relieved by the descent of a gall or intestinal stone, or foreign body, or by the coming away of a polyp, or, in especially fortunate cases, even of an intussusceptum itself, and pressure upon the intestine by tumors can be relieved by their spontaneous or mechanical reduction in size, or by a rectification of position (*e. g.*, in retroflexion of the uterus, ovarian cysts, etc). Even cicatricial stenosis may, in very rare cases, when the period of advancing contraction is over, again become soft; indentations and retractions of the intestines by the products of chronic peritonitis, may, in a similar manner, for example, by the rupture or stretching of false ligaments, be wholly or in part removed. Passages that have been obstructed by neoplasms may again become free by their softening and ulcerative destruction. Stenoses of the lower end of the intestine are often accessible to successful surgical treatment. Finally, an incomplete cure, lasting perhaps for years, may be obtained by the spontaneous or surgical creation of an anus praeternaturalis.

Though the prognosis also varies with these circumstances and depends upon the nature of the constricting process in each case, yet it must be considered in general as a very unfavorable one, both quoad vitam and quoad valetudinem completam.

The duration of constriction of the intestine is chronic, but is in the highest degree variable and dependent upon a number of factors, some of which likewise influence the prognosis materially. It depends upon the anatomical process (*e. g.*, malignant tumor—simple cicatricial stenosis), upon the physical condition of the contraction (*e. g.*, its narrowness and length, whether an annular stenosis in the axis of the intestine, or an angular indentation and retraction, whether smooth-edged or valvular), it also depends upon the rapidity of the origin and growth, upon the completeness and permanency of the compensating hypertrophy of the muscular coat, and, finally, upon the seat of the constriction and its course (peritonitis, perforation, formation of an anus præternaturalis, etc.). We shall return to this subject when speaking of the different anatomical kinds of constriction.

Diagnosis.

The diagnosis follows from the above-described train of symptoms. It presents no difficulty if the stenosis can be directly made out by examination of the rectum, or if its well-marked symptoms are associated with the signs of a chronic (simple, carcinomatous, tuberculous) peritonitis, or with a tumor that can be felt through the abdomen or vagina. In other cases, the history and the consideration of earlier diseases supply important diagnostic points. Among these may be mentioned a previous dysentery or existing tuberculosis, the fact that a strangulated external hernia once existed, and that after its reduction the symptoms of constriction of the intestine gradually appeared, or the knowledge of a previous typhlitis, peritonitis, of the swallowing of a foreign body, the passage of an intussuscepted portion, fæcal stones, etc. The diagnosis of an existing constriction is easy, if the symptoms of internal strangulation first appear and are then followed by those of diminished permeability. Neither is there any difficulty in the diagnosis when the gradually appearing signs of constriction of the intestine are associated with increasing carcinomatous cachexia and the presence of a painful tumor in the abdomen, and when the age of the patient is compatible with malignant new-formations.

On the other hand, we must not forget that occlusions of the intestines often remain latent for a long time, or present only very slight symptoms, so that in the absence of any information furnished by examination, or by the history of the case, the diagnosis is utterly impossible. It should not surprise us if the existence of an occlusion of the intestine is ultimately demonstrated in patients who have long used the most various remedies for a so-called colica flatulenta, habitual constipation, hypochondria, hysterical tympanites, atony of the bowels, or chronic intestinal catarrh. The symptoms of this affection are by no means so characteristic that they cannot likewise be produced by other diseases, such as tuberculosis of the intestine, ulceration of its mucous membrane, chronic or subacute peritonitis, chronic intestinal catarrh, etc.

The symptoms of constriction of the intestines are often nothing but those of impeded advance of the contents, and this may depend as well upon a real constriction as upon functional causes or deficient power of propulsion. So long as important anamnestic or definitive objective testimony is not to be had, the diagnosis of constriction of the intestines cannot be made with certainty, and the physician is much more likely to be right if he considers the impeded advance and evacuation of the contents of the intestine to be due to other functional causes.

As I have repeatedly spoken of the diagnostic information which examination of the rectum may furnish us in many cases of occlusion and constriction of the intestines, it seems proper that we should study this subject rather more closely.

Stenosis of the rectum can usually be detected by introducing through the anus one or more fingers, previously besmeared with oil. The examination should be made with the patient upon his side, or in the knee and elbow position. It is often desirable to have the patient strain, as if at stool, so as to bring the constricted point within reach. The inexperienced may mistake hemorrhoids, or the often rather rigid folds of mucous membrane projecting into the lumen of the rectum, for stenosis, not to mention those rare cases (especially in old people) in which the examining finger may enter the narrow neck of a diverticle, or those in which a firmly lodged plug of fæces has been taken for organic obstruction.

If the digital examination furnishes no information, the higher portions of the rectum must be examined by means of the sound, or by forcible manual exploration. The rectal sound can give reliable information only when used by a careful and experienced hand. For this purpose we use elastic bougies, or, still better, a not too thin œsophageal tube, with a knob-like end, which has been placed for a few moments in warm water and then oiled. The operation is greatly facilitated by injecting warm water through the tube, while it is being pressed upwards, by means of a syringe connected with it. This frees the passage and distends the walls of the rectum and colon, which are so full of folds. When an obstruction is met the sound must be drawn back a little and another attempt made, while more water is pumped in, to pass it by what is often merely an accidental obstruction. It is often well to introduce one or two fingers beside the sound to correct any faulty direction it may have. The sound is still more likely to go astray than the finger is. It often catches in one of the plicæ sigmoideæ, or is arrested by the sphincter ani tertius or superior (Nélaton's sphincter), which is situated five inches above the anus, or by the promontory. Sometimes the sound, after some resistance, passes suddenly and apparently easily upwards, but in fact it remains in the so-called rectal ampulla, lying between Nélaton's and the lower sphincter, having become bent upon itself several times, as can often be seen after its withdrawal. Finally, it may happen that the sound passes upwards through a moderately tight stenosis, or between an intussusceptum and its sheath, and then the physician supposes the rectum to be free at a point where, on the contrary, the obstacle is situated.

The distance to which the sound can be passed depends upon the position of the rectum, and especially of the sigmoid flexure. Although O. Beirne thought it was never passed further than into the flexura iliaca, while Rilliet,[1] and afterwards Cadge,[2] showed by experiments upon the cadaver that it never passed through the descending colon, but at the most only reached it or the sigmoid flexure, others believed that in a few cases they had succeeded in passing the sound one hundred and sixty-six, or even one hundred and ninety-six centimetres (Wachsmuth),[3] and thus to have passed over a corresponding length of the intestine, to have wound through the flexure and sounded the entire colon—the length of which is from four to seven feet—as far as the cæcum. Cadge, and afterwards Simon,[4] explained this errror. In those cases they had to deal with a sigmoid flexure arranged, as is not unfrequently the case, in a large loop, and rendered movable by a long mesocolon. When the sound, after having been engaged in this, is pushed further along, it raises it up, stretching the meso-colon sometimes as far as to the bend of the ribs on the right side. The sound can there be felt through the abdominal walls, and thus give rise to the erroneous belief that it has been passed up to the transverse colon.

The diagnostic value of an examination with the sound must be considered as a subordinate one.[1] Most stenoses of the rectum can be reached with one or two fingers. But when the obstruction is seated higher up, in the region of the sphincter tertius or the sigmoid flexure, it cannot usually be determined, when the sound is arrested by some insurmountable obstacle, whether it has reached the physiological limit of its advance, or whether it has been stopped by a pathological or accidental obstruction. Only repeated examinations, always yielding the same result, and the sense of touch which often allows us to recognize that the sound is passing with difficulty through a constriction, and that, on being withdrawn, it suddenly slips out with a jerk, occasionally furnish valuable information.

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[1] Mem. sur l'invagination chez les enfants. Gaz. des hôp. 1852.

[2] British Med. Journ. 1868.

[3] Virch. Arch. Bd. XXIII. 1862.

[4] Archiv f. klin. Chir. von Langenbeck. 15. Bd. 1873. S. 122.

Recently the upper portions of the rectum, and even part of the rectal end of the sigmoid flexure, have become accessible to direct palpation by the method of introducing the whole hand through the anus into the rectum. To Simon,[2] of Heidelberg, belongs the credit of having methodically described and confirmed and perfected by numerous investigations, this mode of examination which I saw Nussbaum employ in his Clinic, about the year 1865, and which Maunder,[3] in 1868, used for the diagnosis of rectal stenosis. The bloodless method of this manual exploration consists in the introduction of the whole hand through the gradually distended anus during deep chloroform narcosis. Great narrowness of the outlet of the pelvis, especially in men, or an unusually large hand on the part of the examiner, may interfere with the practicability of this mode of exploration. In other cases the introduction of the hand is aided by nicking the cutaneous border of the anus in several places, and, in exceptional cases, division of the sphincter backwards in the raphe is necessary. The resulting incontinence of fæces lasts ten to twelve days (Simon). After the hand has been introduced, three or even four fingers can be carried still further up into the highest part of the rectum, through Nélaton's sphincter, and a short distance into the sigmoid flexure, and by raising up the latter, which is allowed to move by its mesocolon, it is possible, under favorable circumstances, to reach the region of the umbilicus, as I saw Nussbaum do, and even the xiphoid process. In this way different organs in the abdominal cavity can be felt through the sigmoid flexure. The method of this manual exploration, which is always made under chloroform, appears to be without danger when carefully done—a certain amount of force is unavoidable; but yet it is by no means an indifferent operation, and it should be made use of only when there is actual need of it, when important diagnostic information can, with a great degree of probability, be expected from it, or therapeutical ends attained, as in the case of a foreign body, before the performance of laparotomy or colotomy, etc.

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[1] The same is true of examination with a rectal mirror, or with a broad vaginal speculum arranged for the purpose. But still these can be advantageously employed to render visible a colon-, or ileo-cæcal-invagination, that has been forced down into the rectum, or a tumor, polyp, foreign body, etc., and may sometimes have a diagnostic and therapeutic value.

[2] Transaction of the First Congress d. deutsch. Gesellschaft f. Chirurg. Berlin. 1872; and Langenbeck's Archiv f. klin. Chirur. 1873. 15. Bd. S. 99.

[3] Lancet. 1868. Med. Times. 1869.

We have, finally, to mention another means of determining the location of the constriction or occlusion—that is, the injections employed by Amussat and Monro with this view, which aim to show the capacity of that portion of the intestine which lies below the obstruction. The diagnostic value of this mode of examination is very limited. Only when the obstruction is situated very low in the rectum can its presence be inferred from the fact that injections of water, as often as they are used, flow or are in a measure driven immediately out again with considerable force, and that often after even a little water has been injected the further working of the pump is notably resisted. On the other hand, the possibility of injecting large quantities of liquid shows the absence of occlusion, although not of constriction of the rectum or lower part of the colon. This is liable to lead to many mistakes.[1] The accidental obstruction of the rectum by a hardened mass of fæces, the contraction of Nélaton's sphincter, can produce the same results as a stenosis or occlusion. Moreover, numerous examples have shown that still larger quantities of water can be injected through a constriction which presents an insurmountable obstacle to the fæcal masses coming from above downwards, because its upper orifice is covered by a valve-like fold of mucous membrane.[2] The capacity of the intestine for injections varies extraordinarily in healthy people, and all attempts to draw fixed rules for determining the position of the obstruction from the amount of water that can be injected have been unsuccessful.[3] The amount that can be injected and the promptness with which it is again evacuated depend upon a number of variable factors in each case, as, for example, upon the distensibility of the intestine, its condition of contraction (for it is often greatly contracted below the stenosis), upon the amount of the intra-abdominal pressure, and also, other things being equal, upon the quantity of gaseous or solid contents present at the time, upon the length and position of the colon, etc.

Moreover, the rapidity with which injections penetrate the intestine varies greatly, as experiments upon the cadaver show. While small amounts of liquid are often sufficient—when the colon is moderately contracted and the abdominal pressure slight—to reach the cæcum, in other cases greater opposition is made to its advance, and it is only after the lower part of the colon and the rectal ampulla have been distended by the injection of large quantities that the liquid makes its way into the transverse and descending colon. The position of the patient is an important factor: for when he lies upon the right side, the passage of the liquid into the cæcum is facilitated; when upon the left side, it often becomes impossible.[1] We shall again take up the effect of injections when considering the therapeutical results that can be obtained by means of them.

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[1] Thus Cadge (British Med. Journ. 1868) performed colotomy in a case in which the sound could be introduced only six inches, and injected water flowed immediately out again. The autopsy showed a false ligament strangulating the ileum.

[2] *E. g.*, *Watson*, Edinburg Med. Journ. 1871. *Nélaton*, L'Union. 1857. 89. Brit. Med. Journ. 1858.

[3] Brinton, for example, laid down the following rule: if 500 cubic ctmrs. of liquid can be injected, the obstruction cannot be lower than the upper end of the rectum; if 1,500 cubic ctmrs. can be injected, it cannot be lower than the upper end of the sigmoid flexure. In a case of occlusion of the ascending colon 4½ litres were injected. Any one can easily convince himself by a few experiments upon the cadaver how unreliable these estimates are.

Examination per vaginam may furnish important diagnostic information by showing the existence of a retro- or anteflexion, of a tumor arising in the genital organs or in the wall of the pelvis, of a vaginal hernia, etc.

Now that we have discussed the pathology of occlusions and constrictions of the intestine, we shall take up the consideration of their different anatomical varieties and their special symptomatic and other peculiarities.

Internal Strangulation of the Intestine.

Strangulatio s. Incarceratio interna. Etranglement interne. Innere Darm-Einklemmung.

Strangulation by Peritoneal False-ligaments.

These are the products and remains of a previous peritonitis, and unite the most diverse points of the abdominal and pelvic walls with the organs and sides of these cavities. These are represented by fine and tense, sometimes "cartilaginous," or thicker strings and bands, or broad membranous expansions. Peritoneal adhesions are drawn out into long ligaments, in consequence of the changing relations of the abdomen, especially when one end is attached to a movable loop of the intestines. The way in which false ligaments lead to internal strangulation varies greatly, and I select from a full record lying before me the following frequently recurring main varieties:

1. *Strangulation under the false ligament.*

Formation of bridges or arcades under which intestinal loops become engaged and strangulated.

This occurs especially with short bands tightly stretched along some firm surface, such as the posterior abdominal wall, or especially that of the pelvis, the right iliac fossa, uterus, etc. Strangulations of this kind are often caused by ligaments which attach the two parallel sides of a loop, that has been directed vertically upwards by a peritonitic process, and bridge over the corresponding portion of the mesentery. If a loop of intestine becomes incarcerated under this bridge the bowel becomes closed at four points simultaneously.[1] Or a false ligament runs transversely to the axis of the intestine, from the free border of the ileum to its mesentery, bridging over the point of union between the intestine and mesentery. If an intestinal loop becomes incarcerated under this bridge the bowel becomes closed at three points simultaneously. Incarceration is also not unfrequently caused by false ligaments stretched along the mesentery;[2] finally, strangulation is very frequently caused by ligaments running from the cæcum or vermiform appendix to the peritoneum of the iliac fossa.

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[1] *Hall*, Monthly Journal. 1846.

2. *Strangulation by simple loops or nooses.*

This requires long ligaments, which at times at least are loose, such as are found mainly in the abdominal cavity, in the narrower sense of the term, where the relations of the parts are such as to allow of their formation.

Many seem to believe that the simple snaring of the intestine in the loop of a false ligament can take place without difficulty, so long as there is a suitable band. But whoever tries the experiment upon the cadaver, with artificial ligaments, soon convinces himself of the extraordinary facility with which the normally movable intestine slips out of such rings. If we examine more closely, we find that in most cases of simple snaring the strangulated part of the intestine has previously possessed certain peculiarities by which the occurrence of this kind of strangulation has been rendered possible. Thus we find the ends of the loop brought together, sometimes very closely, at its base, in consequence of chronic mesenterial peritonitis, so that it is distinct from the adjoining loops and stands out from the mesentery in a condition similar to that in which we find the sigmoid flexure when it has been twisted upon itself. It is evident that with this unusual fixation of its base the loop must possess abnormal mobility.

The snaring of a loop thus altered occurs in different ways. The lax band may form a ring or spiral between its fixed points *a* and *b* (Fig. 1) in which the intestinal loop becomes accidentally engaged, and then strangulated, generally in consequence of unsuitable attempts [on the part of nature] to liberate it (especially by the addition of a second loop to the incarcerated first one[3]). Or a

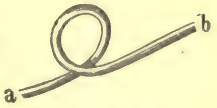

Fig. 1.

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[1] *E. g.*, *Rokitansky*, l. c. Abbild. by Froriep, l. c. *Marshall Hall*, in Astley Cooper, l. c. *Abercrombie*, l. c., p. 151. Gazette Médicale, 1852. *Druitt*, Med chir. Transact. Vol. XXX. 245.

[2] *E. g.*, Catalogue of St. Barthol. Hosp. No. 19. *Hilton*, *Golding-Bird*, Med. Chir. Trans. Vol. XXX. 1847, 1. *Fagge*, l. c. *Concato*, Virch.-Hirsch Jahresb. 1871.

[3] Examples of this kind are given by *Rokitansky*, l. c. Abbildung by Froriep, l. c. *Contague*, Gaz. méd. de Paris, 1853, p. 717. *Curling*, Lond. med. Gaz. Dec. 1846. *Monro*, l. c.; and many others.

loop of intestine, altered as above described, falls over the false ligament, twists around and under it, and then straightening itself out, brings the previously straight ligament into a strangulating spiral, processes which can be easily demonstrated artificially. If this process is repeated, a double loop is formed.[1] In this way Vidal (l. c.) saw the whole small intestine included in a double loop about its mesentery and strangulated. Since the first incarceration excites very active peristaltic motions, it sometimes happens that the rest of the band forms a second noose. The false ligament takes the figure-of-eight shape with strangulation of two or even several intestinal convolutions.[2] Sometimes the incarcerated loop is repeatedly twisted about its mesentery at the point where it is crossed by the ligament, and thus the strangulation is increased. In other cases the incarcerated loop becomes bent at the strangulated point, in consequence of the meteorism, and its top is dragged down into the true pelvis.

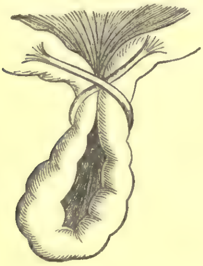
FIG. 2.

Another kind of strangulation which belongs here is that in which a false ligament, attached at one or two points to the intestine, forms a ring through which the whole of the intestine included between the two points of attachment of the ligament passes.[3] (Fig. 2.)

3. *Strangulation by formation of a simple knot* is different from simple snaring.

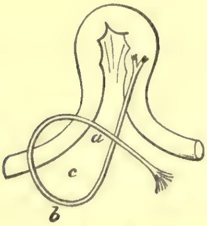

FIG. 3.

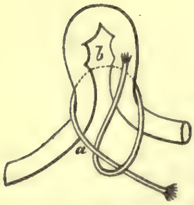

FIG. 4.

Cases of this kind are often described inexactly in the records and not unfrequently misunderstood. There are several kinds of this knotting. The most frequent is the following. The long and loose ligament is fastened at one end to a loop of the small intestine and hangs in the form of a simple coil (Fig. 3). If the top of the intestinal loop passes directly through the coil *c*, a simple knot is formed about the piece of the intestine, as shown in Fig. 4. It is evident that the same result can be produced by the coil being thrown over the top of and around the intestinal loop. Another and rarer kind of knot is made as follows: a long and perfectly loose false ligament forms a simple coil, like that shown in Fig. 1, between its points of attachment *a* and *b*. If now one leg of the so-formed primary noose passes through it we have a knot like that shown in Fig. 5, and if now the intestinal loop passes directly through *c* (Fig. 5), it becomes firmly caught and strangulated (weaver's knot. Nodus textorius). Knots made by false ligaments after the fashion of those known in surgery as "ansa simplex,"[1] are also met with.[2] A common characteristic of all described knots is that when the strangulated intestine is freed the ligament can immediately be drawn out straight.

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[1] *Lebidois*, Arch. gén. de méd. 1. Sér. XIII. p. 230, and others.

[2] *Dalrymple* in A. Cooper, l. c. Mus. of Boston, No. 495, mentioned by Duchaussoy and others.

[3] *Bristowe*, Lond. Path. Soc. XXI. *Küttner*, Virch. Arch. Bd. 43.

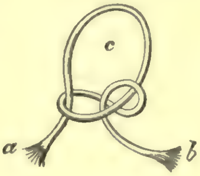

FIG. 5.

4. A false ligament splits into two or more bands, thus forming *buttonhole-like* or wider *spaces* which catch and strangulate the intestine.

This kind of strangulation occurs especially in broader band-like false membranes. Sometimes the ligament spreads out at its point of attachment like a fork or a goose's foot, with several slits between the bands leading to the different attachments.[3] In other cases a slit that is capable of strangulating the intestine is formed between a false ligament and some normal fold of the peritoneum along which it runs. This has been seen at the edge of the Lig. uteri latum,[4] along the Lig. vesicorectale,[5] and also along the free border of the plica mesenterico-mesocolica.[6]

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[1] See *Troschel*, Chir. Verbandlehre. Taf. XI. 8.

[2] Besides the above-mentioned well-determined kinds of knots, others are sometimes described which are quite impossible and depend upon an erroneous apprehension of the anatomical relations. The records of these cases are often injured by confused and unskilful descriptions.

[3] Examples of kind are given bp *Maunoury*, l. c., and in Dict. des Sciences Med. XXIII. p. 569. *Brun*, Bull. de la Soc. Anat. 1834. *Röser*, Journ. f. Chirur. u. Augenh. v. Walter und Amon. Bd. 2.

[4] *Hutchinson*, Med. Times. 1858.

[5] *Guéneau de Mussy*, Arch. gén. de Méd. 1. Sér. T. XXVI. p. 258. *Rokitansky*, Lehrb. d path. Anat. Bd. III. 3. Aufl. S. 190. *Klebs* (Hdb. d. path. Anat. 2. Lief. S. 212) also saw in one case the excavatio rectovesicalis divided into two compartments by an abnormally developed fold of the peritoneum, which projected into the middle, in such a way that one of them was small enough to grasp a loop of the small intestine and easily strangulate it.

[6] This occasionally sharp-edged duplicature of the peritoneum, so named by *W. Gruber*, runs from the lower end of the root of the mesentery of the small intestine to the meso-colon of the sigmoid flexure. The fold is much too loose, when its development is normal, to catch the small intestine and produce internal strangulation. But since I have studied more closely the anatomical relations of the intestines and mesentery, I have seen a case in which the free edge of this duplicature was thickened like a fiddle-string, and separated by a sort of split from the rest of the mesenterial double-fold. I believe I am not wrong in thinking that the internal strangulation, minutely described by Habershon, l. c., p. 479, was one produced by the above-described, probably congenital mesenterial slit in the Lig. mesenterico-mesocolicum. Probably M. Fleury's case, mentioned in the Arch. gén. 2. Sér. T. XV. p. 102, belongs here also.

5. Finally, we have to mention *acute kinking caused by traction*, as an exceptionally rare kind of occlusion of the intestine by means of false ligaments.

This has only been observed in cases in which the false ligament was attached to portions of the ileum which, like the uppermost and lowest portions of the small intestine, have a short mesentery. Thus, Louis (l. c.) saw symptoms of internal incarceration appear after puncture of an ovarian cyst. A false ligament connecting the cyst and the lowest part of the ileum was so stretched by the falling-in of the thick-walled cyst that it bent and closed the ileum by drawing upon it.

Warren (l. c.) saw a pedunculated subperitoneal fibroid of the uterus so wedged in, in consequence of a sudden change of position, between the wall of the pelvis and a false ligament stretched from the lowest part of the ileum to the uterus, that the former was bent and occluded by the traction of the band attached to it. Heller[1] saw a loop of the small intestine that had become adherent to the gravid uterus bent by the traction and occluded when involution of the uterus took place after delivery. The case mentioned by W. Gruber[2] also belongs here.

After incarceration by false ligaments comes naturally *incarceration of the intestine in slits or rings produced by the immediate adhesion of organs in the abdominal or pelvic cavities to one another or to the sides of these cavities.*

Such rings and slits are formed when, for example, the free end of a large appendix epiploicus unites with an adjoining one,[3] with the intestine, the pelvic or abdominal wall, or with the uterus,[4] or when the upturned Fallopian tube[5] becomes adherent to the latter. The forked division which sometimes takes place at the foot of an appendix epiploicus also furnishes rings capable of causing strangulation. This is more frequently produced by intestinal loops themselves which have united with the uterus, the abdominal or pelvic wall (especially in the neighborhood of a hernial ring) or with other loops,[6] in such a manner that slits and rings are left between the adhesions in which another intestinal loop becomes incarcerated.

As for the relative frequency of the above-described various processes of strangulation, that of strangulation under the ligament is greater than all the others.[1] While short, tightly-stretched bands act in this way almost without exception, the formation of nooses and knots requires longer and looser ligaments. For this reason the latter are found almost exclusively in the abdominal cavity ; while, on the other hand, strangulation by bridges and arcades takes place there too, but especially in the cavity of the pelvis. Furthermore, while only a very small intestinal loop is involved in strangulation under a false ligament, in cases of formation of nooses longer intestinal loops (up to five feet, Eppinger, l. c.), or even whole bunches of them, become incarcerated. This is clearly shown by statistics.

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[1] Würtemb. Zeitschr. f. Chir. I. 1851.

[2] Petersb. Med. Ztg. 1861.

[3] *Holmes*, Transac. of Path. Soc. of London. XI. 111.

[4] *Chvostek*, Oestrr. Zeitschr. f. prakt. Heilk. 1867.

[5] *Dorilas*, Giorn. di Med. T. I. p. 91.

[6] *Rokitansky*, l. c. *Macpherson*, Glasgow Med. Journ. 1867. *Abercrombie*, l. c. p. 161. *Herrmann*, Petersb. Med. Zeitschr. X. 3. *Eppinger*, Prag. Viertelj. 1873.

It is commonly asserted, and sought to be proved by insufficient statistics, that incarceration by false ligaments is much more frequent in women than in men. There is supposed to be a reason for this in the greater frequency of peritonitic processes arising in the female organs of generation. Fuller statistics do not support this theory ; but they do show distinctly what differences are due to sex. It is true that peritoneal adhesions and false ligaments in the pelvis unquestionably occur more frequently in women, and we find that incarceration by false ligaments in the cavity of the pelvis occurs four times as frequently in women as in men. But this excess on the side of the women is in part made up for, as the following figures show,[2] by the fact that strangulation by false ligaments in the abdominal cavity (including the upper pelvis) is more frequent among men than among women. To this must be added incarceration by false ligaments starting from herniæ or the adjoining parts, which is also more frequent among men than among women. The frequency of strangulation by false ligaments is thus shown to be about the same in both sexes.

Incarceration by false ligaments occurs most frequently in persons between twenty and thirty years of age, but still quite often in those who are older. Before the age of twenty, on the

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[1] My statistics (thus far the largest) give the following relations :

Strangulation under the band	59 cases.
Formation of knots and nooses	26 "
Strangulation in a slit in the false ligament	7 "
Occlusion by traction	4 "
	96 cases.

[2]

Strangulation by peritoneal false ligaments in the abdominal cavity	35 males.	23 females.
Strangulation by peritoneal false ligaments in the pelvic cavity	8 "	33 "
Strangulation by false ligaments having their origin in hernia	9 "	3 "
	52 males.	59 females.

other hand, and especially in early childhood, it is exceedingly rare.[1] In the three exceptional cases included in my statistics in which children under five years of age died in consequence of incarceration of the intestine by false ligaments, the bands were congenital and probably the products and remains of fœtal peritonitis.

As a rule, the portion of intestine strangulated by false ligaments is the ileum. A fact, which is of importance in incarceration by false ligaments and also in those to be hereafter described, is that the strangulated intestinal loop has often been twisted about its mesenterial axis at the point of strangulation in such a way that the two sides of the loop cross each other.

In a large number of incarcerations of this kind the subsequent distention by gas is of importance in this way, that it often makes the ligament still tenser and the strangulation complete. Sometimes the strangulating band becomes inflamed, gangrenous, and finally ruptures. Recovery as a result of this can occur but seldom, for the intestine must already have suffered too severely before the tough band had become gangrenous. A linear mark is left at the strangulated point in these cases of rupture of the false ligament.

The course of these kinds of internal strangulation is ordinarily acute. The fatal end has often been reached in ten,[2] sixteen, or twenty-four hours, in other cases only after several days. In ninety-six cases the average duration until death was six days. On the average, strangulation by a noose has a more rapid course than strangulation under a false ligament. The beginning of the affection is usually as acute as its course, and not unfrequently occurs during perfect health. In other and not less numerous cases the sudden appearance of the symptoms of strangulation is preceded by prodromal diarrhœas or a constipation lasting for several days, sometimes by prolonged chronic abdominal disturbances depending upon chronic peritonitis, or by the signs of constriction of the intestine. Sometimes the acute beginning is followed by partial restoration of permeability. Under such circumstances the period of acute strangulation is succeeded by that of chronic or incomplete incarceration.[3]

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[1] I cannot give the statistical details here.

[2] *Reveridge*, Brit. Med. Journ. 1868.—*Hauston*, Neue Samml. auserl. Abhdlg. Bd. XV. St. 2.—*Coutenot*, Gaz. des hôp. 1852, p. 439; and many others.

[3] Aertzl. Ber. d. k. k. allg. Krankenh. in Wien. 1860. S. 163.

Strangulation by the Omentum.

Strangulation by the omentum is always accomplished by the aid of previously formed adhesions. There are various ways in which this is done :

1. *Omental ligaments.* These produce strangulation in the ways already described in full under the head of false ligaments.

Sometimes the entire omentum is rolled up into a single string which is adherent and imprisons the intestine.[1] More often portions of the omentum have separated in the form of ligaments, especially from its lateral borders. Adhesion always precedes the formation of such isolated cords. The fretting of the omentum thus induced leads to thickening of the adherent portions. These become ligamentous and gradually separate from the rest of the omentum. This process of separation is favored by the peristaltic action of the intestines.

The omentum is especially liable to form adhesions in old hernias, and the strangulation of the contents of a hernial sac between the omentum and the wall of the sac is an incident well known to surgeons.[2]

2. *Slits in the omentum* cause strangulation comparatively often.

Sometimes the omentum splits up into numerous ligaments, which, as in a case of Wagner's,[3] attach themselves to different points of the abdominal and pelvic cavities and cause manifold strangulations. The adherent portion of an omentum especially divides up into fork-like processes which include narrow spaces between them.[4]

As for the frequency of the different modes of strangulation by the omentum, the annexed statistics will furnish data.[5] It has been asserted, partly upon theoretical grounds, partly upon

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[1] *E. g.*, *Hare*, Lond. Path. Soc. Vol. III. 111.

[2] For details see *Bourillon*, Gaz. des hôp. 1860. *Arnaud*, Mém. de chir. T. II. p. 588. *A. Cooper*, l. c. *Streubel*, l. c. *Gosselin*, l. c.

[3] Oesterr. Med. Jahrb. IV. Bd. 1833.

[4] *Bourbier*, Clinique de Dupuytren, T. III. p. 638. *Peacock*, London. Path. Soc. I. 97. *Wulff*, Petersburg. Med. Ztg. 1869; and many others.

[5] Strangulation under the omental cord	20 cases.
" by slits in the omentum	22 "
" by nooses formed by omental ligaments	9 "
	51

those claimed for false ligaments, and partly upon the strength of insufficient statistics, that these processes occurred more frequently in women than in men. My larger collection gives forty-three males to fifteen females. The greater frequency of omental adhesions in the pelvis of the female is again overbalanced by the greater frequency with which omental adhesions with hernia and the surrounding parts occur in men.

As for the age, course, and all other relations, we can only repeat what was said about strangulation by false ligaments.

Strangulation by the Mesentery.

1. *Slits* and *holes* in the mesentery.

In the majority of cases these are found in the mesentery of the ileum and most frequently at the lowest part of the ileum, especially in the neighborhood of the cæcum. They are rarely in the jejunum, and most rarely in the mesentery of the middle of the ileum. In exceptional cases, strangulation has occurred in slits in the transverse and descending meso-colon,[1] or in the mesentery of the appendix vermiformis.[2] Nothing definite is known as to the way in which these mesenterial openings are formed. Many of them are certainly congenital (Rokitansky; compare also page 529, note 6), others acquired. Of the latter, injuries are often, and sometimes probably not erroneously, supposed to be the cause.[3] Strangulation by these openings occurs either in the ordinary way, or by the formation of knots and kinks, the origin of which it usually is not difficult to explain.[4]

2. Strangulation *under a tense and fixed mesenterial band.*

If a loop of the ileum becomes attached to any suitable point, in a large external hernia or to the female genital organs, for example, the corresponding part of the mesentery becomes sometimes tightly stretched along the posterior wall of the abdomen and the brim of the pelvis. An intestinal loop may now pass between the tense and fixed mesentery and the posterior abdominal or pelvic wall. I know of only four recorded cases in which this has been observed.

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[1] *De Haën*, Rat. Med. T. XI. C. III. Tab. 2. *Brambilla*, quoted by Voigtel, Handb. d. path. Anat. S. 567. *Rembolt*, Oesterr. Zeitschr. f. pract. Heilk. No. 6. 1865. *Davies*, Edin. Journ. 1845. *Löbl*, Oesterr. Zeitschr. d. Ges. d. Aerzte in Wien. 1841. I. 1. 154.

[2] *Partridge*, Lond. Path. Soc. Vol. XII. 110.

[3] *E. g.*, *Roser*, Journ. f. Chir. u. Augenheilk. von Walther und Amon. Bd. 2.

[4] I refer to *Albers*, Atlas d. path. Anat. Tab. XXI. Text IV. Abth. I., and Casper's Wochenschrift. 1837. *Rokitansky*, Oesterer. med. Jahrb. X. 1836. *Taylor's* Brit. Med. Journ. 1871.

Strangulation in Slits and Holes in Different Organs and Parts of Organs.

Of this rare occurrence I have to mention: strangulation of the intestine in a slit in the suspensory ligament of the liver (Barth[1]), in the broad ligament of the uterus (Ertl,[2] Quain,[3] and Bericht des Wiener allg. Krankenh. 1863. S. 80), also strangulation by a tear in the uterus (Percy[4]), tear in the bladder (Cloquet[5]), strangulation by a double perforation of the intestine (Schüppel[6]), or by perforation of the rectum (Brodie,[7] W. Gruber,[8] Stein,[9] Adelmann[10]). Finally, four cases belong here in which an intestinal loop entered a hole made in the peritoneum by an injury or during an operation and became strangulated there (18 Jahrb. d. deutschen ärztl. Ver. in Petersb. 1836–1837, Reich[11], Guillot and Chassaignac[12].

Strangulation by Diverticles.[13]

Anatomy.—The real, or Meckel's, diverticle is found only in the small intestine, at an average, although variable, distance of from one to three feet above the ileocæcal valve.[14] It always arises from the part of the intestine opposite the insertion of the mesentery, and is composed of all the layers with the general characteristics of the small intestine. Its length averages from two to four inches, but varies between that of a blunt peg-like projection (Räsfeld) and ten inches (Dexpers[1]). It is usually narrower than the intestine, and is given off from it, sometimes at a right angle, sometimes at a more or less acute one. Its form is most frequently cylindrical with a conical end.[2] It is sometimes bent, constricted, pouched, or supplied with one or more knob-like swellings—secondary false diverticles. The diverticle often has a small sickle-shaped mesentery, starting from the mesentery of the intestine, and carrying a vein and an artery arising from the superior mesenteric artery. The end of the diverticle is usually free, though it may be attached to the mesentery, the small intestine, cæcum, pelvic organs, anterior abdominal wall in the linea alba under the navel, or to the latter itself.[3]

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[1] Schmidt's Jahrb. Bd. 96. S. 207.

[2] Allg. Wien. med. Ztg. 1864.

[3] Lond. Path. Soc. XII. 103.

[4] Dict. des sciences med. Art.: Ruptures.

[5] Mus. Dupuytren. No. 182.

[6] Arch. d. Heilk. Bd. VII. 1866. 167.

[7] *Brodie*, Lancet. 1827.

[8] Virch. Arch. XXXII. 2. Heft. 205.

[9] Schmidt's Jahrb. 84. Bd. S. 306.

[10] Prag. Viertelj. 1863. 2. 42.

[11] Schmidt's Jahrb. Bd. 110. S. 202.

[12] Both quoted by Duchaussoy, l. c.

[13] *Meckel*, Handb. d. path. Anat. 1. 553, and in Reil's and Autenrieth's Arch. IX. Heft. III. *Sandifort*, Obs. anat. Path. Lugd. Bat. 1777. L. I. C. X. *Gualth v. Döveren*, Passio iliaca subito lethalis. Spec. obs. Acad. Groning. et Lugd. Bat. 1765. C. 5. § 4. p. 79. *Falk*, De ileo e diverticulis. Diss. Inaug. Berol. 1835. *Schröder*, Dissert. Inaug. Erlangen. 1854. *Froriep's* Chir. Kupfert. 343. Weimar. 1836. Heft. 68. *Monro*, Morb. Anat. 1811. Tab. 20. p. 539. *Eschricht*, Müller's Arch. f. Anat. Phys. etc. 1834. *W. Gruber*, Petersb. Med. Ztschr. I. 33. *John Struthers* Anat. and Phys. Observ. I. 1854. 157. *Cazin*, Arch. gén. 6. Ser. 1. p. 475. 1863. *M. Parise*, Gaz. des hôp. 1851. *Malgaigne*, Bull. de l'Acad. de Med. T. XVI. p. 373. T. XVII. p. 28. *Räsfeld*, Diss. Inaug. Berlin. 1852. *Joh. Wagner*, Med. Jahrb. d. österr. St. IV. Bd. 1833.

[14] In exceptional cases this distance is still greater. Thus in the London Museum there is a real diverticle situated fifty-four inches above the cæcum; another preparation (No. 1819, ^{50}) shows a diverticle in the middle of the ileum. William Major (Lancet, Vol. I. 1840.) describes most minutely a diverticle on the jejunum, and in the Aerztl. Bericht. des. allg. Krankenhauses in Wien for the year 1862 (p. 221), mention is made of a real diverticle seven inches long, situated at the junction of the jejunum and ileum. Such an abnormally high diverticle can moreover be understood by the aid of embryology. If for any reason the development of the anterior arm of the middle intestinal loop is checked the greater part of the small intestine is formed by the posterior or lower arm. In such a case the diverticle must be abnormally high. (Consult Embryology.)

A stout band, called the ligamentum terminale (the obliterated vasa omphalomesenterica), sometimes arises from the free end of the diverticle or laterally from its body, and is adherent to some point in the abdominal or pelvic cavities, or sometimes floats freely between the intestinal convolutions, and, exceptionally, is split into two or more bands. This band is often hollow for a short distance from the free end of the diverticle.

We must now mention certain congenital ligaments[4] which are most closely connected genetically with the formation of diverticles, and frequently cause internal strangulation. I consider it desirable to discuss this circumstance with some detail, because the origin of the ligaments is entirely misunderstood, and they are usually looked upon erroneously as peritoneal false ligaments. As is well known, the real intestinal diverticle is due to the ductus omphalomesaraicus remaining patent. (For details, consult books on Embryology.[5]) It may happen, that while the ductus is completely obliterated down to the ileum and thus no diverticle can be formed, its obliterated vessels may remain in the form of a solid cord attached to that point of the ileum where diverticles are usually found.[6] If the cord, which originally stretches from the navel to the lower part of the ileum and can likewise remain in that position,[7] breaks loose from the navel, the end that thus becomes free may, under suitable conditions, become again attached at any point of the abdomen; and in this way we meet with ligaments which, starting from a point on the ileum, from one to three inches above the ileo-cæcal valve, are inserted into the mesentery, into any part of the small intestine, or into the pelvic viscera, etc.

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[1] Réc. de mém. de méd. milit. 1867.

[2] In two cases a pancreas accessorium has been found at the free end of an intestinal diverticle, a formation as interesting as it is difficult to explain. (Zenker, Neumann, Wagner's Arch. d. Heilk. XI. 1870.)

[3] In such a case the diverticle may be hollow up to the navel. *Beck*, Münchner illustr. Med Ztg. II. Bd. Prager anat. Mus. No. 479 and 865. *Meckel*, l. c., *N. Ward*, Path. Soc. of London. VII. 205.

[4] The theory, so frequently met with, that many congenital ligaments in the abdominal cavity are the remains of amniotic (!) cords, shows complete ignorance of embryology.

[5] *Kölliker*, Entwicklungsgeschichte. S. 363.

[6] *Schröder*, l. c.; *Wagner*, l. c.; *Savopoulo*, l. c.

[7] *Wilks*, Trans. of Path. Soc. of London, XVI. 126.

The ligament may, however, become separated at its point of insertion, and the end that is thus made free may attach itself anywhere. We then find cords which start from the navel (or the abdominal wall immediately below it), and extend to some point of the abdominal cavity.[1] These cords are sometimes hollow for a certain distance[2] (Wilks, l. c.). Finally, I must also make mention of other congenital ligaments hitherto misunderstood, which also have a most close genetic connection with the formation of a diverticle. These are cords which start from the mesentery very close to the intestine, at the usual height at which diverticles are given off, and run upwards towards the root of the mesentery, to which they become attached. These ligaments, starting from the mesentery, and naturally never found hollow, are nothing else than the obliterated vasa omphalomesenterica, not the portion lying between the intestine and the navel, but that included between the intestine and the main blood-vessels in the root of the mesentery. A beautiful preparation, made by Dursy, convinced me that the vessels running along the ductus omphalomesentericus from the vitellus represent, after passing the point where they cross the middle intestinal loop to reach the root of the mesentery, an isolated cord (*E* in Fig. 6,) between which and the mesentery a bristle (Fig. 6. probe *S*) can be freely passed along the whole length of the cord. This disposition may remain, as examples show, until later periods of life.[3] Sometimes the ductus omphalomesentericus is obliterated as usual, and nothing shows the connection between the embryonal middle intestine and the vitellus except the above-described peritoneal cord (Fig. 6 *E*) representing the vasa omphalomesenterica between the small intestine and the root of the mesentery, which remains as a ligament, between which and the mesentery the intestine may become strangulated. The accompanying diagrammatic figure needs no further explanation; it appeared to me to be necessary to the understanding of the foregoing. Each one of the three parts, *L* (Lig.

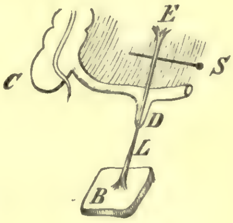

FIG. 6.

B, Abdominal wall in the neighborhood of the navel. The rest is explained in the text. *C*, Cæcum.

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[1] *Spangenberg*, Arch. f. Phys. v. Meckel. V. Bd. S. 87.

[2] Several cases are recorded in which the diverticle was hollow up to the navel (to which it adhered), and after the umbilical cord was divided, fæces were evacuated through it, sometimes with simultaneous prolapse of the diverticle in the form of a red tumor of mucous membrane. (*Siebold* quoted by Schröder, l. c. Also *Dubois*, Bull. de la Soc. anat. 1838. *W. King*, Guy's Hosp. Rep. 1843. *Broadbent*, Path. Soc. of London. XVII. *Webner*, Dissert, Halae 1854. *Gesenius*, Journ. f. Kinderh. I. z. 1858. Lond. Univ. Coll. Hosp. Mus. No. 5347.)

[3] *Falk*, l. c. with a good picture. *Coronel*, Schmidt's Jahrb. 1857. Bd. 96. 210.

terminale), *D* (Diverticle), and *E* may alone persist, or all three may be present together [1] (Falk).

The kinds of strangulation caused by diverticles are essentially the same as those caused by false ligaments.

1. Strangulation *under the diverticle* or its terminal band. This is by far the most frequent.

The free end of the diverticle is especially likely to unite with the intestine or with the mesentery, and thus form a ring through which an intestinal loop may pass and become grasped. Cases are not rare, like those described by Rokitansky and pictured by Froriep, in which the terminal band of the diverticle becomes attached to a higher point on the ileum, and the whole of the convolution lying between the diverticle and the insertion of the terminal band becomes strangulated under the latter (Fig. 7).

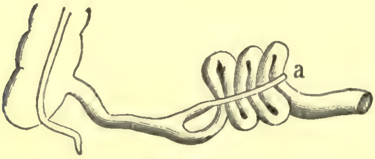

FIG. 7.
a, Insertion of the Lig. terminale in the mesentery of the ileum.

2. The *long loose ligamentum terminale* of the diverticle gives rise to the formation of *loops* and *knots* in exactly the same way as that already described in detail under the head of false ligaments.

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[1] The diverticulum spurium is an acquired formation, caused by the hernial projection of the intestinal mucous membrane through a gap in the muscular coat ("hernie tunicaire" of Cruveilhier). This false diverticle is composed only of mucous membrane and peritoneum, and almost always arises at the insertion of the mesentery into the intestine, and makes its way between the two layers of the mesentery, separating them from each other. It occurs in the descending and lower horizontal duodenum, more rarely in the jejunum, and most frequently in the large intestine, especially in the sigmoid flexure and the rectum, usually in old people suffering habitually from constipation. Diverticula spuria are sometimes formed above constrictions of the intestine (Morgagni). In the large intestine they are often present in large numbers and of different sizes, scattered all along the insertion of its mesentery. Alibert counted two hundred in the colon, and Schroeder's and A. Cooper's fine plates, as well as Bristowe's and Sidney Jones's pictures (Lond Path. Soc. Vol. VI. 191, Vol. X. 132), testify to the number of such false diverticles. Fiedler describes diverticles of the large intestine composed of all the tunics (Denkschr. d. Gesellsch. f. Natur. u. Heilk. Dresden. 1868). Strangulation by false diverticles is as rare as it is common by the real ones. (*Fano*, Union méd. 1849, p. 238.) On the other hand, the latter may lead to formation of concretions with their consequences, to ulceration, and in this way to peritonitis perforativa.

3. Occlusion at the point of origin of the diverticle by traction.

This occurs in the following way: the diverticle attached to the abdominal wall by a ligamentum terminale becomes so stretched, in consequence of meteorism, that the lower part of the small intestine (fastened down by a short mesentery) is drawn upon and bent at the origin of the diverticle to such an extent that its lumen is obliterated (Falk, Wilks, l. c.). Occlusion by traction also may occur when meteoristic intestinal loops become engaged under the terminal ligament, and, although not themselves strangulated, stretch the ligament to such an extent that the lumen of the intestine becomes closed at the point of origin of the diverticle. In both cases the obstruction of the intestine increases the meteorism, and this in turn increases the traction.

4. Strangulation by real "*diverticular knots*" ("anse diverticulaire").

We have already said that the terminal ligament of the diverticle may form nooses and knots in exactly the same way as false ligaments do. Only a free diverticle, one without a terminal band, can make a knot. It is noticeable that straight diverticles, with a pouch-like dilatation of the free end, form these knots with especial frequency, and one might be led to consider the swelling of the free end as secondary to the tying of the knot, were it not that this form is also frequently found when there has been no strangulation; we must, therefore, rather believe that on the contrary this dilatation of the end of the diverticle favors the formation of a knot. The first kind of strangulation is the one that results when the free diverticle coils around a loop of the small intestine and ties a real knot. Before the incarceration takes place the diverticle forms a ring into which its own free end projects. (Fig. 8.) If now an intestinal loop passes from below, *a*, through *c*, it carries the pouched end along and ties the knot. Or the intestine passes from above, *b*, through *c*, pushing the pouched end to one side, and then, on attempting to return, draws it along with it, and thus prevents its own escape by tying the knot. The anatomical specimen always shows which of these two processes has taken place, for, in the first case, the strangulated loop and the pouched loop are on the same side, and, in the second case, they are upon opposite sides of the ring. Still, for many knots of this kind the possibility of an active participation by the diverticle cannot well be precluded.

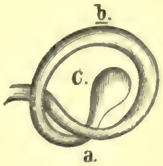

FIG. 8.

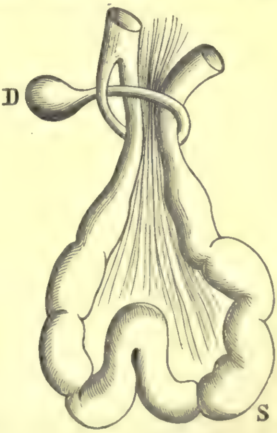

FIG. 9.

Another way in which the diverticle can form a knot is that in which the knot is made, not by the diverticle alone, but in connection with the intestinal loop from which it arises. The accompanying figure, representing a knot of this kind (Fig. 9), renders a detailed description unnecessary.

The comparative frequency of the different kinds of strangulation by diverticles above described is shown by the annexed figures.[1]

Diverticular strangulation occurs much more frequently in men than in women. Of my statistics of sixty-six cases, fifty-two were in males, fourteen in females.

It also occurs most frequently between the ages of twenty and thirty years,[2] and is likewise very frequent between the ages of ten and twenty. It has also been seen at earlier ages, and I know of eight cases between the ages of two and ten, and one, the youngest, at the age of eight months.[3] After the age of forty, diverticular strangulation is exceedingly rare. I find only two cases recorded in which the age was more than thirty, one of them being forty-eight years old, and the other described by Eschricht (l. c.) as " an old woman." The portion of intestine incarcerated by the diverticle is, without exception, the ileum, as in the other kinds of strangulation above described.

The course of this kind of strangulation, especially that due to the tying of a knot, usually leads promptly to a fatal result, often in from twelve to twenty-four hours. The average duration of this kind is four days, of strangulation under the diverticle six days.

Strangulation by the Appendix Vermiformis.

The vermiform appendix repeats the same processes of strangulation that we have seen caused by diverticles. According as its free end becomes attached to the cæcum, ileum, ovary, etc., bridges, rings, and arches are formed, which may lead to strangulation. Sometimes the appendix is wound in the form of a

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[1] Strangulation *under* the diverticle or its terminal band........ 40 cases.

[1] Strangulation *under* the diverticle or its terminal band........	40 cases.
By nooses or knots formed by the ligamentum terminale.......	14 "
Diverticular knots..	12 "
Acute occlusion by traction................................	4 "
	—
	70 cases.

[2] Fagge's opinion, that diverticles are found more frequently in people under than in those over the age of twenty, is not supported by fuller statistics. The average age of seventy cases was twenty-five years.

[3] *Trier*, Pfaff's Mittheil. Jahrg. III. Heft. 9.

close spiral—"instar cornu arietini" (Ruysch)—or of a snail-shell, or in that of a ring, as shown in Fig. 8. If now an intestinal loop makes its way into it, it either is imprisoned in the spirals (which it often draws out still longer), or it transforms the ring, as was described when we were speaking of diverticles, into a real strangulating knot of the appendix.[1] The comparative frequency of the different kinds of strangulation is shown by the annexed figures.[2]

The statement has been everywhere accepted, upon the strength of insufficient statistics, that strangulation by the vermiform appendix occurs much more frequently in women than in men, because the relatively frequent pelvic peritonitis of the former more often gives occasion for the formation of adhesions. Admitting this to be so, it is yet completely overbalanced by the greater frequency of inflammation and consequent adhesions of the appendix in males. Taking the figures of Crisp, Volz, and Crouzet together, I find in one hundred and six cases of inflammation of the vermiform appendix and typhlitis, eighty-nine males and seventeen females. Corresponding to this, of thirty-four cases of strangulation by the appendix, twenty-one were males, thirteen females. What was true of the age with reference to false ligaments is also true here, with the addition that cases occur still more rarely in early life. The youngest person mentioned in my statistics was fifteen years old. The average age was forty years. The strangulated portion of the intestine was always the ileum, with one single exception, when it was the cæcum. The course and duration are the same as in strangulation by the diverticle.

Strangulation by Internal Hernia.

If, starting from a purely practical standpoint, we designate as *internal hernias* all those "in which no tumor visible externally appears," many cases of hernia obturatoria, perinealis, ischiatica, of interstitial, inguinal, or crural hernia would have to

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[1] Recorded examples: *W. Gruber*, Med. Ztg. Russlands. 1860. Nos. 14–19. *Al. Monro*, Essays of a Soc. of Edinb. T II. *Pfeiffer*, De Laparotomia, etc., l. c. *Blöst*, Bayer. med. Corresp - Blatt. 1845. *Fagge*, l. c. *Bennet*, London Path. Soc. IV. 146. *Raimbert*, Bull de la soc. anat. 1837. *Lallemand*, Clinique de Dupuytren. T. III. 635. *Rostan*, Arch. gén. de méd. XIX. 1. Sér.; and many others.

[2]

Strangulation under the adherent appendix..................	28 cases.
" by the appendix drawn out in a spiral..........	4 "
" by the formation of a knot.....................	4 "
	36 cases.

be included. Anatomically speaking, we draw the line correctly when we include among the internal hernias those which lie entirely within the abdominal or thoracic cavities (hernia bursæ omentalis, diaphragmatica, intermesenterica, interepiploica), or which, as sub- or retro-peritoneal, lie parallel to the abdominal wall and project into the abdominal cavity, without ever making their way outwards, even when their size increases. In contradistinction to these, external hernias are those which the action of the diaphragm forces outwards, and which, as they increase in size, always appear as tumors which can be detected upon the outside.

We shall first speak of those external hernias which sometimes exactly resemble internal strangulation—that is, which present no hernial swelling and no special symptoms indicating the anatomical cause of the incarceration.

1. We have to mention that each of the usual external hernias (inguinal, femoral, ventral) can be of such small size (hernia incompleta, incipiens) that they betray themselves, even to the most skilful observer, by no signs. This is especially true of femoral hernia in fat people; but it has also repeatedly occurred in hernias of the linea alba, of Gimbernat's ligament, and more rarely in inguinal hernia.

2. Those interstitial (intraparietal, intermuscular) hernias, standing, as it were, between the external and internal hernias, such as arise at all hernial openings,[1] but most frequently in the inguinal canal, sometimes present the symptoms of internal strangulation. When they enter these openings they follow at first the usual direction towards the exterior, and then turn and pass interstitially between the muscles and fascia of the abdominal wall. These hernias may appear alone or in connection with an ordinary inguinal or femoral hernia, representing a collateral projection, a pocket in the hernial sac. In the latter case they are usually the result of previous attempts at reduction or taxis. Strangulation of an interstitial pocket of a hernial sac has been repeatedly seen associated with complete mobility of the main hernia.

3. Finally, the following hernias, which, with the exception

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[1] Hernia juxta-inguinalis, juxta-cruralis, juxta-umbilicalis (Cruveilhier).

of H. foraminis ovalis, are very rare, sometimes resemble "internal hernia." We mention them briefly, referring to surgical text-books for the details:

a. Hernia foraminis ovalis s. obturatoria:[1] Tumor may be absent; pressure upon the region of the foramen ovale is painful; Romberg's "femoral pain," consisting of formication and painful tension along the adductors of the thigh; four times as common in women as in men; has never been known to occur before the age of thirty, and in most cases happened after the age of forty.

b. Hernia ischiatica passes through the sciatic notch on the upper border of the M. pyriformis; usually forms no tumor until the hernia, increasing under the glutæus, makes its appearance. By pressure upon the overlying sciatic nerve it causes sciatic neuralgia.

c. H. perinæalis, H. labii majoris postica s. pudendi.

d. H. rectalis and H. vaginalis: Complications of prolapse of the rectum or vagina.

e. H. lumbalis is found in Jean Louis Petit's triangle, the base of which is formed by the crest of the ileum, its inner border by the sacro-lumbalis muscle, and the outer border by the M. obliquus abdominis externus. The triangle is covered by the deep layer of the fascia lumbo-dorsalis, through which the hernia makes its way.[2]

Let us now take up in order the different "*internal hernias.*"

Hernia Retro-peritonealis Anterior (*Klebs*),

designated by the French, Hernia intrailiaca, antevesicalis, "Hernie intrapelvienne."

The hernias included under the common name of hernia retro-peritonealis anterior are in fact *subperitoneal herniał sacs* which have their orifice in or close by the internal inguinal ring, from which they spread in different directions, upwards along the ileo-psoas or iliacus muscle (H. intrailiaca[3]), or inwards, towards the bladder (H. antevesicalis[1]), or downwards, behind the horizontal ramus of the pubis into the true pelvis (H. retropubica, "H. intrapelvienne"[2]).

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[1] Thus, John Hilton (Med. Chir. Transact. Bd. 31. 1848. p. 323) performed laparotomy, thinking he had to deal with an internal strangulation, and he found a hernia obturatoria, which he reduced from within the abdomen. Exactly the same thing happened to Walter Coulson (London Lancet. Vol. II. p. 303. 1863).

[2] *Larrey*, Bull. de l'acad. de méd. 1869.

[3] *Quesnoy*, Arch. gén. de méd. Ser. VI. T. 21. *Wagner*, Oesterr. med. Jahrb. IV. 1833. *Dittrich*, Prag. Viertelj. 1845. *Lucke*, Lond. Med. Gaz. 1850. p. 458. *Hanau*, Diss. Inaug. Giessen. 1870; and others.

Sometimes one of these internal hernias is accompanied by no additional rupture; sometimes an inguinal hernia exists simultaneously, and the subperitoneal internal one represents only a pocket of the other. In both cases the hernia retroperitonealis anterior has been supposed to be secondary, because, when it existed with inguinal hernia, it was thought to be a subperitoneal pocket of the hernial sac, caused by repeated attempts at reduction; and, when there was no inguinal hernia present, as the result of the reduction in mass (réduction en bloc) of a previously-existing one.[3] Others,[4] basing their opinions upon good grounds (for example, upon the occurrence of a H. retro-peritonealis anterior in individuals who have never had an external hernia), have maintained that an anterior retro-peritoneal hernia may arise primarily—that is, without any preceding inguinal rupture. Still, it occurs most frequently as the result of réduction en bloc, for the hernial sac of a strangulated inguinal hernia can, by violent[5] taxis, be loosened at all points of its periphery, especially at the opening, and forced, with its contents, into the retro-peritoneal layer between the peritoneum and the fascia transversalis. If, as many surgeons do, we class anterior retro-peritoneal hernias among the "interstitial hernias," it is, at least, proper to distinguish them by the name "internal interstitial" from those external ones in which the hernial sac lies between the different muscles or between them and the fascia transversalis.

Among the anterior retro-peritoneal hernias we include likewise the two following internal hernias, which occupy the iliac fossa, and which may also be called intrailiacæ.

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[1] *Leneveu*, Bull. de la soc. anat. 1837. p. 289. *Hernu*, Recueil périod. de la soc. de méd. de Paris. T. XI. 291. *Arnaud*, On hernia, pars II. Sect. II. p. 387. Obs. 9; and others.

[2] *Cooper Forster*, Guy's Hosp. Rep. X. p. 143. *Sanson*, Dict. de méd. et de chir. Tom. IX. Art. Hernie. *Streubel*, Schmidt's Jahrb. Bd. 110. S. 362. *Paris*, Sur deux variétés nouvelles de hernie. Mém. de la soc. de. chir. T. II. p. 417.

[3] *Gosselin*, Leçons sur les hern. abdom., recueillies par Léon Labbé. Paris, 1865, p. 23. *Streubel*, Ueber die Scheinreduct. Leipzig. 1864. *Arnaud*, On hernia. l. c.

[4] *M. Paris*, l. c. *Faucon*, Arch. gén. de méd. 1873. VI. 21. Compare *Dittel*, Wien. med. Wochenschr. XVIII. 7. 1862. The largest H. retroper. ant. known to me is the one described by Lehmann, Schmidt's Jahrb. Bd. 118. S. 119. It is at least doubtful whether Eberth's case (Hufeland's Journ. 1840. IV.) belongs in this category.

[5] Dupuytren (Leçons de clin. chir. T. V. p. 549. Ed. 2. 1839) accepts this exclusively. Others believe that, if the predisposition exists, even moderate attempts at taxis are sufficient to cause reduction in mass. *Servier*, Mém. cour. de l'occlusion intestinale. Liège. 1870. *Streubel*, Verhdlg. d. med. Gesellsch. z. Leipzig. 1864. Bd. I. *Linhart*, Vorles. über Unterleibsbrüche. Würzburg. 1866.

Hernia Interna Vaginalis Testiculi.[1]

Hitherto this has been seen only in cryptorchids.

As is well known, the processus vaginalis peritonæi arises as an independent extension of the peritoneum into the scrotum, at a time when the testicles are still within the abdominal cavity. The descent of the latter is brought about by the gubernaculum Hunteri, and the direction of the traction is determined by the plica gubernatrix which projects into the vaginal extension. Under certain circumstances it happens that the processus vaginalis does not extend into the scrotum, sometimes not even into the inguinal canal, but remains in the iliac fossa as a peritoneal pouch. The descent of the testicle is arrested either in the iliac fossa or at the abdominal ring of the inguinal canal. In other cases the processus vaginalis appears not to have been formed.[2] The testicle, drawn by its guiding band, makes a fold of the peritoneum behind it; but, on account of the abnormal resistance, it does not pass beyond the internal abdominal ring, and thus forms a peritoneal pouch lying upon the iliacus or psoas muscle—our H. interna vaginalis testiculi. Finally, a normal vaginal extension may extend into the scrotum, and yet the testicle, when drawn downwards, may, by being abnormally attached to the peritoneum, draw it after itself, and thus form a second pouch. Under these circumstances the testicle usually descends only into the inguinal canal, but occasionally even into the scrotum. In this way, I think, can be explained many of the double hernial sacs found in congenital hernia.[3]

The second kind of internal hernia which we include among anterior retroperitoneal hernias is the one to which attention was called by Biesiadecki.[4]

Hernia Iliaco-Subfascialis.

The peritoneum of the iliac fossa is continued through an opening in the iliac fascia between the latter and the iliacus muscle, and thus forms a hernial sac, the mouth of which looks upwards. The origin of this fossa iliaco-subfascialis is explained by the supposition that the tendon of the psoas minor, which has a fan-shaped attachment to the iliac fascia, happens to be unusually strong, and forms a fold projecting over the latter, the edge of which is afterwards the orifice of the hernia. The descending colon has formed part of the contents of the sac in all the cases thus far observed.

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[1] The term H. externa vaginalis testiculi is applied to the congenital inguinal hernia which results from the processus vaginalis remaining open.

[2] Perhaps because the abdominal and pelvic walls have grown too rapidly as compared with the retarded development of the peritoneum.

[3] I refer, for the details of this subject, which in part is still under discussion, to the special surgical treatises; also to *Faucon*, Arch. gén. de méd. 1873. VI. 21. *Bourguet*, Gaz. hebd. 1864. No. 44. *Fagès*, Rec. de la soc. de méd. de Paris. T. VII. p. 38–40. Faucon and Broca believe that many cases of H. interna vaginalis testiculi, with cryptorchidism, were originally external inguinal hernias, which were reduced en bloc with the testicle, during childhood (?). *M. Paris*, Arch. gén. de méd. VI. 6. 96.

[4] Unters. aus. d. path anat. Inst. in Krakau. Wien, 1872 (Braumüller), p. 19, et seq.

Hernia Duodenojejunalis.

Synonyms: "H. retroperitonealis" (Treitz); H. mesocolica (A. Cooper); H. mesogastrica interna (W. Gruber); also known as "H. retroperitonealis posterior;" and H. intermesenterica, intermesocolica.

A. Cooper, The anat. and surg. treatment of abdom. hernia, by Aston Key, London, 1827, with plates.—*Peacock*, Transact. of the Path. Soc. of London. Vol. II. 60, 61, with plates.—*Treitz*, Hern. retroperiton. Prag. 1857. (Credner).—*W. Gruber*, Virch. Arch. Bd. XLIV. *Lambl*, Beob. u. Stud. a. d. Geb. d. path. Anat. Prag. 1860.—*Waldeyer*, Virch. Arch LX.—*Pye-Smith*, Guy's Hosp. Rep. XVI. 1871. *Eppinger*, Prag. Viertelj. 1870. Bd. 108.—*Landzert*, St. Petersb. med. Zeitschr. 1871. pp. 350-383.

This hernia was first described in the year 1776, by Neubauer, in a Jena dissertation; it was studied more closely and beautifully pictured by A. Cooper; but its real significance was first made known, and it was thoroughly investigated—historically as well as anatomically—by Treitz. As a result of the researches of the last-named author, and of the lively interest taken in it by the anatomists (W. Gruber, Waldeyer), we possess a very full and valuable literature upon the subject. We now know of forty-two certain and four probable[1] cases of hernia duodenojejunalis, which completely confirm and add scarcely anything new to the account given by Treitz in his classical work.

Anatomy.—If, upon the cadaver, the great omentum and transverse colon (Fig. 10, *C*) are thrown upwards, and the mass of the small intestine pressed to the right, the left side of the flexura duodenojejunalis (*F*) is exposed encircled by a crescent-shaped fold of peritoneum (*c*), the plica duodenojejunalis, whose concave edge is directed to the right and somewhat upwards, its upper end being continuous with the lower layer of the transverse mesocolon (*m*), and its lower end with the serous covering of the duodenum. The vena mesenterica inferior, on its way to join the splenic vein, passes along the border of the lowest part of the plica; at a variable distance from the edge of the lower horn is the arteria colica sinistra (a branch of the mesenterica inferior) supplying the left part of the colon. Behind the plica is a usually shallow pouch, the fossa duodenojejunalis[1] (Huschke), which, according to its size, contains more or less of the convexity of the flexura duodenojejunalis, and, under certain circumstances, the whole of it, in which case it already represents a hernia. If now more and more of the jejunum and ileum passes into the fossa, its cavity, which normally is funnel-shaped, broadens into the form of a bag and spreads out in the retro-peritoneal cellular tissue, partly raising up the peritoneum parietale posticum, partly stretching it and using it for its hernial sac. The layers of the transverse and descending mesocolon, which ultimately are wholly used in the formation of the pouch, share the same fate; the transverse and descending colon become intimately adherent to the hernial sac, which they almost surround. The hernia can increase in this way to such a size that it comprises the whole of the small intestine and fills the entire square of the colon (Fig. 11). The hernial orifice, meanwhile, gradually enlarges, for the lower horn of the plica duodenojejunalis spreads along the root of the mesentery, often as far as to the right sacro-iliac articulation. The border

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[1] As such are to be reckoned the cases of Hauff, Bordenave, Hesselbach, and Ridge-Hilton.

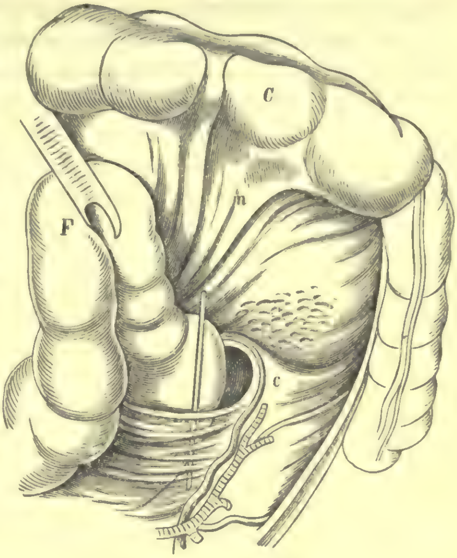

FIG. 10. (After Treitz.)

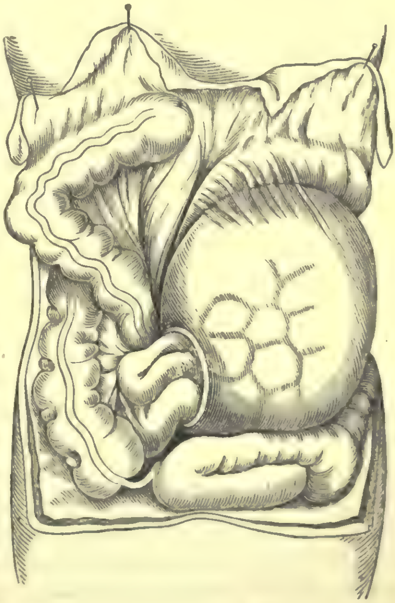

FIG. 11. (After Treitz.)

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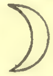
[1] For the interesting embryological details I refer especially to Treitz and Waldeyer, l. c.

of the hernial opening includes the vein and artery above mentioned, forming a stout ring which renders any enlargement of the orifice in that direction impossible.

The form of the plica duodenojejunalis, with its concavity towards the right, and its position on the left of the vertebral column, causes the hernias, almost without exception, to develop towards the left side of the abdomen. But if, under especial circumstances, the crescent of the plica is so arranged that its concavity faces to the left, or if the fossa duodenojejunalis happens to lie to the right of the vertebral column,[1] the development of the hernial sac may exceptionally take place towards the right half of the abdomen (Klob), in which case the parietal peritoneum, between the root of the mesentery and the ascending colon, is drawn upon for the formation of the hernial pouch, and the hernial orifice is removed from the above-mentioned ring of vessels. If the lower horn of the plica opposes, or if the deep diameter of the fossa duodenojejunalis runs obliquely from within and below to the left and upwards, or if the peritoneum parietale posticum cannot be easily pushed back, on account of its intimate adhesions with the posterior wall of the abdomen, the hernia spreads wholly, or mainly, between the layers of the transverse and descending mesocolon (H. mesocolica Cooperi). On the other hand, when the upper horn opposes, or has grown together, or when the deep diameter of the pouch runs more vertically from above downwards, or when the layers of the mesocola offer an insurmountable resistance, the hernia is developed mainly in the retroperitoneal position and the hernial sac is entirely, or in great part, formed of the peritoneum parietale posticum (H. retroperitonealis posterior). Sometimes two pouches exist with two plicæ , of which the outer one, carrying the vessels, circumscribes concentrically the inner one, or the anterior pouch (Waldeyer). Since I have paid more attention to the fossa duodenojejunalis, I have repeatedly met with varieties, especially multiple divisions, by means of secondary folds.[2]

The etiology of H. duodenojejunalis is unknown. It probably is sometimes congenital, occurring in the latter part of fœtal life, or at least it occurs soon after birth. The youngest case was a child two months old, the oldest sixty-five years. This hernia is found more frequently in males than in females, in adults than in children.

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[1] This, as W. Gruber showed, is the case when the position of the duodenum is abnormal, if its lower horizontal portion, before it passes under the transverse mesocolon, turns once more to the right and appears upon the right side of the vertebral column. The course of the whole duodenum then has the form of the letter S. Treitz also noticed these anomalies of position (l. c., pp. 127, 128). They depend, as in Gruber's case also, sometimes upon the fact that the lower branch of the middle embryonal intestine has not appeared over the upper one, and then they usually coincide with a common mesentery for the jejuno-ileum and colon. For the details I refer especially to W. Gruber, l. c. and also in other places.

[2] We certainly cannot approve of the attempt which has recently been made (Landzert) to base a new division of hernia duodenojejunalis upon such varieties.

The tension of the peritoneum, the stretching which it undergoes during the formation of the hernial pouch or when its contents are forcibly disturbed, the many wrenchings which the stomach especially suffers (its tightly stretched ligamentum gastrocolicum often lies spread out over the greater part of the hernial pouch as a third layer of peritoneum when the transverse colon is drawn downwards), the manifold displacements and wrenchings which the colon and mesocolon experience, explain the frequent colics, dyspepsia, coprostasis, and their subjective annoyances, and lead not unfrequently to depression of spirits and sometimes to marked hypochondria. It is very common for peritonitic processes to occur in the neighborhood of the hernial orifice and pouch, and these may give rise to adhesions and lead to stenosis of the ingoing or outcoming intestinal loop, to chronic incarceration and its consequences.

Only in very rare cases does acute strangulation of this hernia take place.[1] Most of the examples were accidentally found during examination of the bodies of the individuals who had died of different diseases.

Under favorable circumstances, if the hernia is of notable size, I consider it possible to make a probable diagnosis, not a positive one, but still one that is based upon reasons. The circumscribed globular distention of the mesogastrium, with retraction of the region corresponding to the colon, the firm, elastic, spherical lump which can be distinctly felt when the abdominal wall is thin, giving the impression of a large, somewhat movable cyst, and extending from the mesogastrium principally to the left, the peculiarity that this well-defined tumor always yields a sonorous note on percussion and clear intestinal sounds on auscultation, also the presence of hemorrhoids and the loss of blood from the rectum in consequence of compression of the inferior mesenteric vein, permit, when taken in connection with the subjective troubles indicating chronic disease of the abdominal organs, a probable diagnosis to be made. In case laparotomy is performed to relieve acute strangulation, profuse bleeding would be caused, when the hernial orifice was enlarged, by the division of the arteria colica sinistra and the vena mesenterica inferior, which latter is usually much dilated.

Internal Hernias in the Neighborhood of the Cæcum.

Various more or less constant peritoneal pouches are found in the neighborhood of the cæcum. Whoever will take the trouble to examine with reference to this point the cadavers that come in his way, will soon be convinced of the extraordinary variability, preventing any uniform nomenclature, of these relations, and it would be as easy as it would be unnecessary to make new descriptions and classifications. Hernia rarely takes place in the region of the cæcum: first, because the pockets formed there are unusually small and shallow, and have the common peculiarity of a comparatively wide orifice and a funnel-shaped continuation; secondly, because the orifices usually are not formed of stout rings, but of soft folds diminishing towards the free border; and, finally, because the pouches are turned away from the small intestine and look downwards.

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[1] In the forty-two certain cases strangulation occurred only three times (Peacock, Bryk, Biagini).

The accompanying purely diagrammatic figure I owe to the kindness of Professor von Luschka.

The *recessus ileocæcalis*, Luschka[1] (fossa ileocæcalis media, Hartmann;[2] fossa ileocæc. inferior, Waldeyer), Fig. 12, probe *b*, is the most constant of all the pockets which occur here and lies between the mesentery of the appendix vermiformis and the plica ileocæcalis.

The *recessus ileocæcalis superior* (Hartmann), Fig. 12, probe *a*, is due to the fact that a light fold of the right layer of the mesentery of the small intestine, carrying a branch of the arteria ileocolica, overlies the junction of the ileum and the cæcum. The pouch thus formed between the fold mentioned and the plica ileocæcalis is usually very shallow and is often lacking.

Fossa ileocæcalis infima, Hartmann (fossa subcæcalis, Treitz, named recessus retro-appendicularis by me), Fig. 12, probe *c*. When the appendix vermiformis is raised directly upwards, its mesentery is stretched, and sometimes a funnel-shaped depression can be seen between the lower layer of the latter and the cæcum. If the finger is laid in it, it seems, when seen from above, to be covered by the mesenteriole and the plica ileocæcalis. The point of this funnel-shaped depression sometimes extends to the pointed or lower angle of the junction between the cæcum and ileum, sometimes the depression is flat and shallow, and often it is absent. Once I saw it divided by a second fold going directly from the appendix to the parietal peritoneum—a sort of second

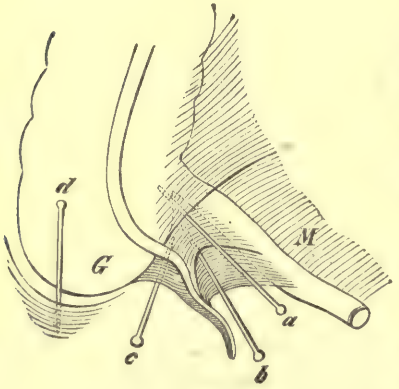

FIG. 12.

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[1] Anat d. Bauches. S. 171; Arch. f. pathol. Anat. 1861. S. 285.

[2] Die Bauchfelltaschen in der Umgebung des Blinddarms. Dissert. præs. Luschka. Tüb. 1870.

mesenteriole—into two shallow chambers, one of which lay between the cæcum and appendix, the other on the inner side between the latter and the real mesentery which carried the arteria and vena vermicularis. It is unquestionably most likely that the internal hernia with strangulation observed by Snow,[1] was in this peritoneal pouch.

Fossa retrocæcalis (F. subcæcalis, Treitz). This lies behind, and is covered by the cæcum, and is first brought into view by raising up the cæcum; in thirty per cent. of the cadavers, according to Waldeyer, we then see a pocket or groove, sometimes shallow, sometimes deep, lying between the layers of the mesocolon ascendens and extending into it, and bounded at its orifice by two parallel borders. The orifice of course is directed downwards. It is the analogue of the fossa intersigmoidea, which is soon to be described.

I know of only three recorded cases of hernia retrocæcalis,[2] two of them with strangulation.

The *fossa infracæcalis* (Fig. 12, probe *d*) lies upon the iliacus muscle (properly is an "intrailiaca"), and forms immediately below the cæcum a pouch, sometimes large, open on the upper side, reaching from the border of the cæcum to the mesenteriole, and looking almost as if intended to receive the former. Sometimes the appendix vermiformis takes its place. Engel[3] saw this pocket also dilated to form the pouch of a hernia, comprising nearly all the small intestine, with simultaneous dislocation of the cæcum to the left and upwards.

Hernia Intersigmoidea.[4]

The fossa sigmoidea (Treitz), first mentioned by Hensing, in 1742, in a Giessener dissertation, lies in the mesentery of the sigmoid flexure. If the latter is raised up, so that its mesentery is made tense, a groove can be seen starting from the left, and extending between the two layers of the mesentery of the sigmoid flexure, with its opening directed downwards. According to Waldeyer and Treitz, it is found in from eighty to eighty-eight per cent. of the bodies examined; according to Gruber, in only sixty per cent. The opening into the fossa is sometimes in the form of a fissure, sometimes closed with a sort of valve, and the fossa itself is sometimes divided up into several parts by secondary folds (Hensing, Waldeyer). Hernias in this groove are exceedingly rare. Lawrence[1] mentions having found one such by accident. The case described and figured by De Haën,[2] and erroneously supposed by many to be an example of this, is nothing but the strangulation of a portion of the intestine in a fissure of the mesocolon of the sigmoid flexure.

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[1] Lond. Med. Gaz. 1846. Treitz, l. c., p. 109, considers this as a case of hernia subcæcalis, to which, in fact, he gives such an extension (p. 107) that my fossa retro-appendicularis as well as the following retrocæcalis would both be included in it. However, Snow's case is not a H. fossæ ileocæcalis, as Hartmann supposes.

[2] *Rieux*, Thèse de Paris, 1853, p. 14. *Escalier* (Th. de Rieux), *Thévenot*, quoted by *Duchaussoy*, l. c., p. 359. Compare as probably belonging here also the case given in the Bericht. d. k. k. allg. Krankenh. in Wien, 1864, p 234.

[3] Wien. med. Wochensch. 1861. No. 40.

[4] I cannot now discuss the details of the embryological genesis of this or of the previously mentioned peritoneal pouches. Compare *Treitz*, l. c.; and *Waldeyer*, Hern. retroperit. Breslau, 1868; and Virch. Arch. Bd. LX.

Hernia Intraepiploica.

The omentum is adherent and rolled up like a horn. A loop of the small intestine sinks down into this funnel and becomes strangulated (Delay[3]). In other cases there has probably been an opening in the omentum. When the intestinal loops made their way through it, they forced the omental layers apart and passed between them (? Baugrand[4]). The latter process can be imagined only in an omentum fixed by adhesions. I suppose, however, that in these cases of internal omental hernia there has been something else involved, which has been overlooked. It has been fully established by the numerous observations of Arnaud,[5] Prescott Hewett,[6] Streubel,[7] and others, that the omentum contained in an inguinal hernia is sometimes wrapped completely about the contents of the hernial sac, and in this way becomes an omental sac, which, as a second internal one, lies within the external inguinal hernial sac. Most cases of H. intraepiploica are previously reduced inguinal ruptures contained in just such an omental sac.

Hernia Ligamenti Uteri Lati.

The surface of the broad ligament of the uterus is depressed at some point of its free border between its two layers, and thus a pouch is formed into which intestinal loops are received. Frequent childbearing, by relaxing the broad ligaments, favors the occurrence of hernias of this kind (Carteron and Saussier[8]).

Hernia Foraminis Winslowii.

Or Hernia Bursæ Omentalis.

The orifice of this hernia is formed by the foramen of Winslow, between the ligamentum hepatoduodenale and the lig. hepatorenale, and may be either round, three-sided, or crescentic. The small intestine has been repeatedly found, and a piece of the colon has been once found, to have passed through this opening into the saccus epiploicus. Strangulation occurred in three recorded cases.[1]

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[1] Abhdlg. von den Brüchen. Translated by Busch, Bremen, 1818, p. 678.

[2] Ratio medendi. Pars XI. p. 141. Tab. II.

[3] and [4] quoted by *Duchaussoy*, p. 357.

[5] Mém. de chir. T. II. p. 588.

[6] Med. Chir. Transact. Vol. IX.

[7] Schmidt's Jahrb. Bd. 110. S. 364.

[8] Annal. de la chir. franc. et étrang. T. VII. p. 257.

Hernia Diaphragmatica.

Morgagni, De caus. et sed. morb. Ep. LIV. Art. 11-13.—*Sömmering*, Ueb. d. Urs. etc. d. Brüche. Frankf. 1811.—*Percy*, Dict. d. sc. méd. Tom. 9. Art. Diaphragma. Paris. 1814.—*Cruveilhier*, Traité d'anat. path. T. I. p. 609.—*A. Cooper*, The anat. and surg. treatment of abd. hernia. London. 1827.—*Duchaussoy*, l. c. —*Reid*, Edinb. Med. and Surg. Journ. Jan. 1840.—*Peacock*, Transact. of the Path. Soc. of London. Vol. XIV. 146.—*Bochdalek*, Prag. Viertelj. 1848. 3. S. 89.—*Bohn*, Königb. med. Jahrb. II. 1859.—*Duguet*, De la hern. diaphr. congénit. Paris. 1866.—*Schrant*, Excerpt. in Schmidt's Jahrb. Bd. 89. S. 169.—*O. Leichtenstern*, Die Diagnose der H. diaph. Berlin. klin. Wochensch. 1874. No. 40, et seq. Also a large number of articles in the journals and of dissertations. Of the latter we may mention: *P. Kirschbaum*, in A. Haller. Disp. chir. select. T. III. LXIX.—*Thurneisen*, Basel. 1777.—*Peters*, Göttingen. 1834.—*Loder*, Jena. 1784.—*Zwanziger*, Halle. 1819.—*Fehleisen*, Tübingen. 1828.—*Dreifus*, Tübingen. 1829.—*Resigius*, Berlin. 1823.—*Butter*, Leipzig. 1849.—*A. O. Fischer*, Berlin. 1840.—*Müser*, Berlin. 1836.—*Würth*, Würzburg. 1847.—*Stierling*, Heidelberg. 1834.—*Weyland*, Jena. 1831.—*Huber*, Basel. 1852; and many others.

The details of my statistics, by far the largest yet collected, of two hundred and fifty-two cases of diaphragmatic hernia, will soon be published in another place.

The name hernia diaphragmatica s. phrenica has been used for a long time to indicate not only those displacements of abdominal viscera into the thoracic cavity in which the organs passing through a hole in the diaphragm possess a hernial sac composed of the peritoneum, or the parietal pleura, or of both of them, but also, in opposition to the strict surgical conception of hernia, all those ectopiæ in which the bowels, in consequence of the existence of a communication between the abdominal and thoracic cavities, lie free within the latter. The first have been distinguished as herniæ veræ, the latter as hernias without a sac, herniæ spuriæ s. falsæ.

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[1] *Jobert*, Traité des malad. chir. du canal intest. T. I. p. 522. *Rokitansky*, Lehrb. d. path. Anat. III. Bd. S. 136. *Blandin*, Anat. topogr. p. 127. Treitz repeatedly saw the jejunum or a loop of the colon loose in the hiatus Winslowii.

The false are far more frequent than the real hernias. Against 212 of the former we have only 28 of the latter.[1] With reference to the location, notable differences exist. Against 180 cases on the left we find only 37 on the right side, a proportion which is nearly the same in congenital and in acquired diaphragmatic hernias.[2] The reason of this must, in a measure, lie in the position of the liver. Congenital defects, more often than the acquired ones, interest large portions of the diaphragm, sometimes an entire half, and often, accompanying actual eventration, are either found associated with other deformities and defects (especially anencephalia, hemicrania), or cause the death of the otherwise well-formed child by asphyxia. Smaller congenital defects with ectopia of a small portion of the intestines can be borne for many years with only moderate discomfort, as positive examples have shown.

There are certain points on the diaphragm which are penetrated more frequently than others. In congenital, and also in acquired hernias, the abnormal opening is found more frequently in the muscular than in the tendinous portion of the diaphragm,[3] a proportion which is especially marked in congenital ectopiæ. The intestines pass much more frequently through the posterior than through the anterior part of the diaphragm. Among the points at which this passage may occur, we have to mention:

1. The *foramen œsophageum* and the muscular and tendinous parts immediately adjoining it.

2. The spot close behind the sternum, pointed out by Morgagni. Between the sternal portion of the diaphragm attached to the processus xiphoideus sterni and the first costal prolongations on each side, attached to the cartilage of the seventh rib, there remain gaps filled with loose, fatty, connective tissue,[4] and under certain circumstances, especially when the sternal portion is lacking, these coalesce and form a single gap. The hernias, often real ones, which occur at this point, pass

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[1] As I have recently seen the occurrence of real diaphragmatic hernia questioned, I will name the authorities for some examples of it: *Fränzel*, Bayer. med. Correspondenzbl. 1846. No. 23. S. 360. *Froriep's* neue Notiz. II. Bd. No. 5. 1837 Sp. 27. *Bonn*, Tab. anat. chir. 1828. Taf 20. *Cruveilhier*, Anat. path. Livr. 17. Pl. 5. *Goblet*, Bull. de la soc. anat. T. XXI. p. 243. *Würth*, l. c. *Textor*, Bayer. Intell.-Bl. No. 20, 1847; and many others.

[2] Against 65 congenital on the left there are 12 on the right.
" 115 acquired " " " 25 " "

[3] Against 74 hernias in the muscular portion of the diaphragm we find 48 in the tendinous. The opening is found very frequently in the muscular near its junction with the tendinous part.

[4] *Luschka*, Anat. d. Bauches. S. 158.

into the anterior mediastinum (named by me H. mediastinica anterior[1]), and usually contain the colon or small intestine, or they make their way towards one or the other, most often the right, pleura. In the same way, fatty[2] and omental hernias may occur at the same place.

3. A similar gap, filled with adipose connective tissue, lies behind between the lumbar and costal portions of the muscle. It is in the form of a triangle, bounded on the inner side by the outer edge of the crus externum of the pars lumbalis, or by the pars intercostalis ima, and on the outer side by the lowest costal prolongation, and whose base is the twelfth rib. This gap, covered by a membrane, and first pointed out by Bochdalek,[3] has furnished, in some cases, a passage for real or false diaphragmatic hernias.[4]

4. Only in isolated cases has the hole through which the sympathetic nerve[5] passes between the crus externum and crus medium served as a hernial orifice. Nor has this ever happened in the foramen pro vena cava or in the hiatus aorticus. Hernias which pass through the hiatus nervi sympathici, or even nearer to the vertebral column, may make their way into and spread out within the posterior mediastinum (Cruveilhier) (H. mediastinica posterior).

The edges of the various large hernial openings in chronic cases of diaphragmatic hernia are rounded, smooth, thickened, and sometimes provided with a few fatty, membranous fragments of the pleura or peritoneum. Adhesions of any extent between the contents of the abdomen and pleura are exceedingly rare, even in chronic cases. Of the abdominal organs, the stomach is most frequently dislodged, then the transverse colon, omentum, small intestine, spleen, liver, pancreas, and kidneys, in the order named.[6] In eighty per cent. of the cases, two or more organs were found at the same time in the pleural chamber. If only one organ was found, it was most often the stomach, next the transverse colon, more rarely the small intestine or omentum. As the communication between the two cavities is usually free and hindered by no adhesions, the contents of the thorax vary with the degree of distention of the intestines. The displaced stomach regularly turns its greater curvature upwards, the smaller downwards. If, under these circumstances, the pylorus remains in place while the fundus is dislodged, the stomach may become twisted about its long axis, and this gives rise to severe symptoms of strangulation.[1]

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[1] *Morgagni*, Ep. 54. Art. 11. *Jagwiz* in *Gohlius*, Art. Med. Berol. Dec. II. Vol. IV. Obs. 1, p. 64. *Bednar*, Krankh. d. Neugeb. Wien. 1852. Würth gives handsome plates, l. c.

[2] *Froriep*, Kupfertafel. 136. 7. *Bérard*, Bull. de la soc. anat. 1826, p. 25.

[3] Prag. Viertelj. V. 3. 1848.

[4] *Elsässer*, Ber üb. d. Gebäranst. in Stuttgart. 1833. *Fischer*, Diss. Berlin. 1840.

[5] *St. André*, Philos. Transact. Vol. 30. No. 351, p. 580.

[6] 136 times the stomach (or part of it).
125 " the colon (transverse).
80 " omentum (still more frequently ; not always specified).
76 " small intestine.
59 " spleen.
30 " liver (portions of left lobe).
24 " pancreas.
2 " kidneys (congenital, through the posterior gap in the diaphragm).

Of the liver, the left lobe is most frequently displaced; in rare cases of H. congenita dextra, fang-like prolongations of it form the contents of a real diaphragmatic hernia.[2]

Diaphragmatic hernia is much more common in males than in females.[3] Certain occupations especially expose to it; thus it is often met with among sailors, soldiers, slaters, carpenters, etc.

Etiology.

Congenital diaphragmatic hernia is due to arrest of the fœtal developmemt. Acquired hernia is more commonly the result of a traumatism, the most frequent being penetrating wounds of the left of the abdomen and thorax, especially stab and gunshot wounds; also falls, usually from a considerable height, and with or without fracture of the ribs (often with fracture of the spine),[4] jarring, and severe crushing.

The opinion, so often repeated in the literature of earlier times, that violent vomiting is the cause of diaphragmatic hernia, had its origin in the fact that when, as is not unfrequently the case, an individual died from diaphragmatic hernia accompanied by constant vomiting, the latter was erroneously supposed to be the cause of the rupture and of the hernia. On the other hand, it is not to be denied that when there is a congenital predisposition, violent vomiting may actually cause diaphragmatic hernia; nor can I doubt those recorded cases in which extraordinary expulsive efforts during delivery have produced rupture of the diaphragm and passage of the abdominal viscera into the thorax.[1] Both facts have been certainly established by undoubted observations. But for the production of a hernia in that way, I believe that there must always be a congenital (or acquired?) predisposition to it on the part of the diaphragm.

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[1] Examples of strangulation of the stomach: *Forlivesi*, Gaz. méd. de Paris. 1843. 2. Sér. T. XI. *Valette* (Delore), Gaz. des hôp. 1852, p. 455. *Spilman*, American Med. Chir. Review. III. 1859. *Smith*, The principl. of forens. Med. London. 1821, etc.

[2] *Engel*, Wien. med. Wochenschr. 1867.

[3] Of 150 cases of acquired diaphragmatic hernia, 128 were in males, 22 in females. Of 65 congenital ones, 35 were males and 30 females.

[4] Laceration of the diaphragm by a fall is decidedly favored by advanced age, as is shown by full statistics. The changes in elasticity may here be taken into consideration. Most of the diaphragmatic hernias that occur in early life are due to stabs or gunshot wounds.

Course, Terminations, Symptoms, and Diagnosis.

It should here be mentioned that real as well as false diaphragmatic hernias are sometimes found by chance in the bodies of those, even aged persons,[2] who have died in consequence of some other disease. If, in cases of acute ectopia due to a traumatism, death is not caused by simultaneous injury done to some vital organ (laceration of the viscera, wounding of the spinal cord, commotio cerebri, hemorrhage, etc.), it may follow the disturbance of the respiration and circulation caused by the compression of the lungs and heart with symptoms of asphyxia, or the strangulation of the stomach or intestine in the fissure made in the diaphragm. In other cases the patients recover from the injury, the hernia becomes chronic and may be borne, often during a long life, either with quite insignificant or with constant symptoms, which will be hereafter described under the head of Diagnosis.[3] If the death is finally due to the hernia, it may happen in different ways: first, by strangulation[1] of the intestine or stomach in the fissure of the diaphragm (in fifteen per cent. of diaphragmatic hernias), especially when it is small; or by acute twisting of the displaced colon[2] about its mesentery. In both cases the incarceration leads sometimes to gangrene and perforation of the displaced intestines. The symptoms of strangulation are almost always called forth by some incidental cause immediately preceding; for example, by an abundant meal, a drinking bout, the operation of an emetic, unusual straining, violent physical efforts, lifting heavy weights, etc. These are the things which sometimes cause sudden death, by producing fatal compression of the lungs and heart, in consequence of distention of the displaced intestines, or of the passage of additional portions into the thorax.[3] Finally, the fatal termination has repeatedly taken place acutely with all the signs of internal strangulation (collapse, violent vomiting, anuria), although such strangulation could not be proved anatomically.

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[1] *Elsässer*, l. c. *Edwards*, Lancet, 1832, and in Behrend's Rep. 1832. *Leroy*, quoted in the Dict. des. sc. méd. T. IX. p. 217. Compare also *Copeman*, Assoc. Journ. March, 1855. It has often been maintained, upon theoretical grounds, that the breaking through of cancers, hydatid tumors, abscesses, empyemas, etc., might cause H. diaphragmatica. I know of only one case of this kind.

[2] For example, a woman seventy-five years old, mentioned by *Cruveilhier*, Anat. path. Livr. 17, Pl. 5; a woman seventy-seven years old, *Lambron*, Gaz. méd. de Paris, 1839, No. 12; a man fifty-five years old, *Billroth*, Langenbeck's Arch. 1869; a man sixty-nine years old, *Dreifus*, l. c.

[3] I mention as examples the following ectopiæ due to traumatisms: My patient (l. c.) died four years after the injury which caused the diaphragmatic hernia. In *Bujalsky's* case (Schmidt's Jahrb. Bd. 77, S. 56) the hernia lasted twelve years; in *Desault's* case

The Diagnosis

of diaphragmatic hernia, made only five times in two hundred and fifty-two cases, is sometimes easy, sometimes difficult, provided the displaced portion of intestine containing air is not too small, and is occasionally impossible. I refer for the details to my article in the Berliner klinischen Wochenschrift, 1874, No.

(Chir. Wahrn. 5. Thl. 145), fifteen years; in Preuss's (Salzb. Ztg. 1798, No. 43, S. 265), *Sargent's* (Boston Med. and Surg. Journ. 1872), and *Michel's* (Würtemb. Correspond.-Bl. 1853, No. 14) cases, twenty years; and in *Grottenschütz's* case (gunshot wound, Medic. Ztg. v. e. V. f. H. in Preussen, 1846), fifty-two years. It is evident that omental or real hernias of the diaphragm give rise to lesions that are hardly worth mention, and can be borne for years without any trouble.

[1] *Larussac*, Kleinert's Report, 1832. *Laurent*, Gaz. des hôp. 1860, p. 451. *Woodworth*, N. Y. Med. Journ. 1874. *v. Gietl*, quoted by Popp, Deutsche Zeitschrift f. Chir. v. Hüter and Lücke. I. Bd.; and many others.

[2] *Goblet*, Bull. de la soc. anat. 1846, p. 248. *Jahn*, Deutsche Klinik, 1867. *Travers*, quoted by Duchaussoy, p. 348; and others.

[3] *E. g.*, my case (l. c.); also *Illjinsky* (Oesterr. Wochenbl. 1842. *Osberghaus* (Casp. Wochenschr. 1837, S. 391). *Heer*, quoted by Morgagni, Ep. 54, Art. 12. *Beck*, Transact. of London Path. Soc. Vol. VI. 220; and others.

40, et seq., and I confine myself here to a short account of the most important points.

The passage of intestines containing air into the thorax gives rise, at points where normally lung is found, to symptoms on auscultation and percussion exactly resembling those of pneumothorax. Usually, therefore, the differential diagnosis is between pneumothorax and diaphragmatic hernia. Both may be the immediate results of a traumatism (stab, shot, contusion, etc.). The pneumothorax ordinarily is complete, and its characteristic signs are to be found at all points of the affected half of the thorax; while diaphragmatic hernia is usually confined to a smaller part of the thorax, and presents the signs of an empty cavity. In other cases the etiology points towards one or the other affection; for example, previous destructive processes in the lungs, an empyema intus perforatum, point towards pneumothorax; and, on the other hand, circumscribed signs of a cavity found long after a traumatism capable of producing a hernia has been inflicted, absence of any pulmonary lesion, and perhaps at the same time indications of internal strangulation, these point towards hernia diaphragmatica. Common to both are: the greater prominence of one-half of the chest, displacement of the heart to the right, absence of respiration on the affected side and of vocal fremitus and the normal respiratory murmurs. Common to both, also, are the percussion note, sometimes drumlike, sometimes more tympanitic, the metallic sound heard on auscultation while percussion is made at the same time upon a pleximetre, and the various metallic-like sounds (gutta cadens, glouglou intestinal). On the other hand, pneumothorax occurs as often on the right as on the left side; traumatic diaphragmatic hernia, with displacement of a portion of intestine containing air, almost exclusively upon the left side. Pneumothorax is absorbed after a while, with expansion of the lung or increase of the exudation; diaphragmatic hernia remains constant, often for years. In pneumothorax the abdomen is greatly swollen, in hernia diaphragmatica it is flat, sometimes retracted, often tense and hard as a board; after violent straining the abdomen, previously distended moderately, remains for some time flattened or even retracted. In pneumothorax the volume of the cavity changes only gradually, and consequently the signs obtained by percussion and auscultation are entirely constant for a long time. Whenever percussion is made upon a pleximetre during auscultation, the metallic sound is always heard, and is always of the same unchanging height over large regions of the thorax. The limits within which the metallic note is obtained remain the same for a long time. Whenever auscultation is practised, the respiration offers a metallic resonance, always with the same pitch; the same metallic murmurs, the same gutta cadens, the same succession phenomena, are always present. In hernia diaphragmatica it is different. There the cavities containing air vary in size, and underlie a sometimes greater, sometimes less extent of the thoracic wall, sometimes contain air, sometimes liquids or solids, are sometimes distended, sometimes contracted, and offer the most various changes in volume and position in consequence of their own peristaltic action. The usual percussion note may therefore be either drumlike, or purely tympanitic, or non-tympanitic, full and sonorous, or muffled or hollow. Percussion upon the pleximetre shows that the region of the metallic sound changes rapidly in extent and outline. Sometimes the metallic note is nowhere to be heard, sometimes it can be obtained over a large surface. At one point the note is high, at another low, and indeed it often changes at the same point during auscultation, runs through the scale, disappears, and returns again immediately. Equally changeable are the metallic sounds heard on auscultation without simultaneous percussion, both with reference to their presence and to their pitch and the extent of their distribution. The various resonant, metallic intestinal sounds that can be heard on auscultation, give sometimes to the hand laid flat upon the chest the sensation of the trickling or crowding by of the contents of the intestine forced along by its peristaltic action. Other signs, and there are many of them, can be obtained by auscultation of the chest while the patient swallows, by careful insufflation of the stomach or colon.

Subjective troubles, also, which in pneumothorax have a certain uniformity, are as different as they are changeable in patients with chronic diaphragmatic hernia. I may mention: pains of different kinds in the abdomen and thorax, dyspnœa often occurring suddenly while the patient is quiet, and then remaining absent for a long time, attacks of suffocation, increase of the dyspnœa after eating, after straining, after physical exertions,—on the contrary, patients with partial ectopia of the stomach feel easier when the stomach is full—sometimes an inability to lie upon one side, in some cases the affected, in others the unaffected one, constipation, symptoms of dysphagia, dyspepsia, incomplete nutrition, hypochondria, etc.

The preceding description of internal hernia is, I think, sufficient for that part of my task which was to deal with displacements of the intestines. I shall hereafter have occasion to speak of the manifold anomalies in the course of the large intestine which are due to an unusually long mesocolon. As for those anomalies of position which are due to the possession of a common mesentery by the jejunum, ileum, and colon, as well as for the transfer of the colon to the left side (due to arrest of the twisting of the embryonal middle intestinal loop), I refer to the sources mentioned below.[1]

It remains now to briefly mention the lateral transposition of the viscera, for it may possess diagnostic interest, *e. g.*, mistaking a liver situated on the left side for a large tumor of the spleen.[2] Transposition of the viscera, in the great majority of cases, affects those of the chest and abdomen at the same time;

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[1] *W. Gruber*, Arch. f. Anat., Physiol. u. wissen. Med. 1862. S. 588. 1864. S. 478. 1865. S. 558. *Treitz*, l. c., S. 125. *Fleischmann*, Leichenöffnungen. Erlangen. 1815.

[2] Virch. Arch. Bd. 22. 1861.

but it is not always complete, all the organs of both cavities do not always change sides. In seventy out of seventy-eight cases the transposition affected both cavities, in the remaining eight it affected only the abdomen.[1] I do not know whether transposition of the thoracic viscera alone (congenital dextrocardia) occurs more frequently than that of the abdominal organs alone, but it seems to me to be doubtful. The situs inversus lateralis occurs more frequently in males than in females, forty-nine to nineteen, according to W. Gruber.

The total transposition of the thoracic and abdominal viscera offers, in very many cases, such definite signs that the diagnosis cannot be difficult for an experienced and attentive examiner. But if the transposition is incomplete and irregular, the diagnosis may be difficult, uncertain, or impossible.

Twisting, Knotting, and Compression.

Twisting of the intestine about its mesenterial axis occurs most frequently at the sigmoid flexure.[2] Its production requires a definite anatomical arrangement of the sigmoid flexure, consisting in this, that its mesenteric root should be unusually small, so that the ends of the loop are brought close together and form a pedicle that can be twisted (Fig. 13, *s*). The greater the disproportion between the length of the S loop and the smallness of its mesenterial root, so much the more easily does it become twisted. The

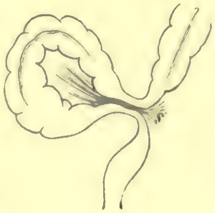
FIG. 13.

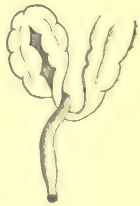
FIG. 14.

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[1] *W. Gruber*, Reichert's and Du Bois Arch. Jahrg. 1865. S. 569, et seq. *Scheele*, Berlin klin. Wochenschr. 1875. Nos. 29 and 30.

[2] My statistics are as follows:

45 twistings of the sigmoid flexure.
23 " " a loop of the ileum.
8 " " the jejunum and ileum combined.
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76

twist may be in either direction, and may extend through an arc of from 180° to 360° (Fig. 14), or it may even be several times repeated.

The anatomical disposition of the S loop above mentioned, as favoring twisting, is often congenital, or is developed in very early childhood. It is well known that the sigmoid flexure is very large in new-born children, and usually extends over to the region of the cæcum.[1] If now the descensus cæci takes place slowly and incompletely, or if the colon grows more rapidly than the abdominal and pelvic walls, it falls into numerous folds, flexures, and curves, which, if mainly limited to the sigmoid flexure, cause its permanent enlargement and abnormal implantation. It is not very uncommon to find this disposition of the sigmoid flexure, very marked at times, in the bodies of young people, without any change or thickening of the mesocolon to show antecedent mesenterial peritonitis. In other cases it is true that a chronic, sometimes fatal, mesenterial peritonitis is the cause of the abnormal implantation of the sigmoid flexure with approach of the end of the loop. This abnormal position of the sigmoid flexure often exists without causing any obstruction to the movement of the fæces. Of this we have frequent enough opportunities to convince ourselves. A short time ago, while examining the body of a boy eleven years old, who had died of pseudoleukæmia and had never had any difficulty in evacuating fæces, I found a chronic twisting of the sigmoid flexure with close approximation of the ends of the loop. If air is forced in from the side of the colon, the S loop untwists, and again resumes its twisted position when the air is allowed to escape, a proceeding that must have been repeated during life with every passage of fæces. If, however, when this disposition of the sigmoid flexure exists, the power of the intestine is weakened by age, or by any other reason in earlier life, it so happens that this twist, which has heretofore been of no importance, now has an actually obstructive influence upon the advance of the fæces. The repeated obstruction of the fæces, together with its mechanical and chemical irritation, the frequent pulling upon the pedicle of the abnormally movable S loop, excite an insidious peritonitis, which leads to thickening and shrinking of its mesenterial root, and thereby to a still closer approximation and constriction of the sides of the loop at their base. The more there is of this, the slower becomes the advance of the fæces through the loop, and the loop itself, like the intestine above a stenosis, often expands to an extraordinary size, in consequence of which the disposition to twisting (*e. g.*, as the result of accumulation of the fæces in one side of the loop alone) is essentially increased.

Impermeability in these cases of twisting is caused by the pressure which the ends of the two sides of the twisted loop exert upon each other. Moreover, as these sides run parallel to the mesenterial axis (see Fig. 13), it is evident that, when the latter is twisted, they, too, must likewise be twisted more or less about their own axes.

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[1] Hence the proposal in the Paris Academy to perform Littre's colotomy upon the right side in new-born children.

But it is not the twisting itself which causes permanent occlusion of the intestine. Other forces are requisite which oppose the undoing of the twist. Among the first of these, in occlusion by simple twisting, we have to mention the often *extraordinary size* and *weight* of the sigmoid flexure, filled with fæces and gas, and extending not unfrequently, under such circumstances, as far as the right hypochondrium, and overlying all the folds of the small intestine. If now such a loop, overdistended and consequently paretic as the result of a constipation that has lasted perhaps for several days, rolls about its axis, its own weight, together with its inactivity, prevents it from straightening itself again. Furthermore, the distention by gas of the intestine above the occluded point, which immediately follows the twisting,[1] and the limitation of the free mobility of the twisted loops which results therefrom, aid in fixing it in its place.[2] Twists of the kind here described often occur after prolonged coprostasis, and make their first appearance as its relatively unimportant final incident, after the severe symptoms of impermeability of the intestine have already appeared, as the result of hyper-extension and paralysis of the sigmoid flexure.

Another and not infrequent process which makes the twist permanent is this, that a portion of the ileum, rendered freely movable by an unusually long mesentery, throws itself across the pedicle of the twisted flexure, and, by the pressure which it exerts, prevents the other from becoming straightened again. The same result can be brought about by peritonitic false ligaments or omental adhesions running directly across the pedicle of the twisted loop and fixing it in its twisted position.[1] The compressing mesentery of the small intestine is often pressed and folded into the form of a cylindrical, stalk-like mass, tightly stretched by the weight of its overloaded convolutions hanging down in the cavity of the pelvis.

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[1] The distention of the colon by gas, after being twisted, is often so considerable, and the diminution of space thereby produced is so great, that it makes a further distention of the small intestine impossible. Under these circumstances, we often find the latter contracted and lying behind, near the vertebral column, covered by the immensely-distended colon and sigmoid flexure.

[2] Examples are given by *Gay*, London Path. Soc. Vol. X. p. 153. *W. Gruber*, Med. Ztg. Russlands. 1860. Nos. 14-19. *Kade*, Petersb. med. Ztg. 1867, p. 167, et seq. *Lingen*, ibid. N. F. I. p. 169. Also by *Rokitansky*, Oesterr. med. Jahrb. X. 1836. *Froriep's* Kupfertafeln. *Küttner*, Virch. Arch. Bd. 43; and many others.

The causes of the lengthening of the mesentery of the small intestine are not always very clear. As a matter of fact, it is found most frequently in advanced life, and when the abdominal walls have become greatly relaxed by repeated child-bearing. In the first case the disappearance of the fat in the mesentery and the other senile tissue-changes play an etiological part. In other cases large hernias, especially the so-called eventration, are the cause of the lengthening of individual portions of the mesentery of the small intestine.

From all this it is evident why occlusion by twisting is not unfrequently preceded for a long period of time by colics, meteorism, especially of the colon, and coprostasis—symptoms due to slight stenosis of the ends of the S loop. Occlusion itself comes on acutely, with the severe symptoms of internal incarceration, rapid collapse, vomiting, excessive meteorism, and usually violent tenesmus. Death sometimes takes place within the first twenty-four hours—on the average on the fourth day. In many cases *frequent*, *sometimes bloody*, *diarrhœa* is observed at the time of or immediately before the appearance of the first symptoms of strangulation, so that invagination of the intestine is often suspected. But it should not be easy to make this mistake, for in intussusception the course is not so rapid, nor the collapse and meteorism so early and severe; and the premonitory intestinal troubles, which, as a rule, last so long in twisting of the intestine, are usually absent in intussusception.

Twisting of the sigmoid flexure occurs most frequently in advanced life,[2] for reasons contained in the foregoing; but the

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[1] *Esau*, Deutsches Arch. f. klin. Med. 16. Bd. S. 474.

[2]

At the age of 10 years	1 case.
From 28 to 40 years	6 cases.
From 45 to 60 years	18 "
Over 60 years	7 "
	32 cases.

anatomical disposition which makes it possible may exist at the earliest age. It is more frequent in males than in females.[1]

In rare, exceptional cases, other parts of the colon besides the sigmoid flexure have been twisted about their mesenterial axis—for example, an abnormally long flexura dextra or sinistra, extending to the mesogastrium or hypogastrium, or the unusually movable ascending colon with the cæcum. The necessary anatomical conditions, as well as the processes which, under those conditions, make the occlusion permanent, are the same as those already described in detail.[2]

Twisting about the mesenterial axis occurs more rarely in the small intestine also than in the sigmoid flexure (compare the statistics on page 561). The anatomical conditions of their occurrence are the same here as there. We distinguish between *twisting of the entire jejunoileum* and *that of single loops or bunches of loops.*

If the root of the mesentery is unusually short, while its height and the length of the intestine are normal; if the radix mesenterii runs more vertically than usual; if the mesentery attains its full height at the jejunum suddenly, and loses it just as abruptly in the neighborhood of the cæcum—then the small intestine is in a condition to undergo twisting as a whole about its mesentery. The twist is usually one hundred and eighty degrees, and the direction such that the upper end of the intestine is carried to the left and downwards, the lower end to the right and upwards. The right side of the mesentery faces to the left, and the left to the right. This twist does not always cause absolute occlusion—often only a constriction at each end of the twisted convolution, the beginning of the jejunum and the end of the ileum, the latter of which, when occlusion takes place, is often twisted at the same time about its own longitudinal axis. Twisting of this kind has been seen in very young children, and it seems as if that variation in the development of the mesentery, in which the ileum, cæcum, and ascending colon possess a common mesentery,[3] especially disposed to it.

Occlusion by twisting of a single section of the small intestine is also rare.[4] The same anatomical disposition is necessary to its production as that which we described for twisting of the sigmoid flexure. Thus, for example, the flexura duodenojejunalis may be unusually high, its two ends approximated by a small mesentery, and twisting take place. Or a loop of small intestine, most frequently of the lower part of the ileum, may become, as it were, set off from the rest by the lengthening of its mesentery and the approximation of its ends to one another, and to the vertebral column as the result of chronic mesenterial peritonitis, and thus a sort of pedicle, that can be twisted, is formed.[1] This condition is found most frequently in the folds of the small intestine, which have lain for a long time in a large hernia, or which have been adherent to the parts about one or to the pelvic viscera.[2] The traction thus produced lengthens the mesentery of the corresponding loop; the repeated pulling upon it sets up chronic mesenterial peritonitis, which draws the ends of the loop still nearer to each other. Twisting of the small intestine leads to permanent occlusion, for the paralytic loop sinks into the true pelvis,[3] and thus produces a sharp bend in itself, or because the pedicle of the twisted part is compressed by another portion of the ileum or by a false ligament, and in this way its untwisting is prevented. Twisting of loops of the small intestine or of the whole jejunoileum occurs more frequently in advanced life and in males. Irregularities in the evacuations of the bowels, symptoms of stenosis of the small intestine not unfrequently precede this accident; profuse diarrhœa is often its immediate occasion.

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[1] Of the thirty-seven cases contained in my statistics, ten were females.

[2] Examples: *Avery*, London Path. Soc. II. 222. *Goupil*, Bull. de la soc. anat. de Paris. 1842. 336.

[3] *Eppinger*, Prag. Viertelj. 1873. I. 1, 56. *Rokitansky*, l. c. Bericht a. d. k. k. allg. Krankenh. in Wien. 1866. S. 143. *Küttner*, l. c. *Esau*, l. c.

[4] We have already mentioned the fact that the intestinal loops which, for any cause (false ligaments, appendix, omental fissures, etc.), become strangulated, often suffer twisting about their mesenterial axis at the same time.

To the consideration of twisting of the intestine naturally succeeds that of the

Intertwining or Knotting of Two Intestinal Loops.[4]

This involves most commonly the sigmoid flexure and a loop of the ileum. It requires a portion of small intestine, rendered unusually movable by a long mesentery, and a long sigmoid flexure with a comparatively narrow mesenterial pedicle. The "knot" may be tied in various ways. It most commonly happens—twelve times in the twenty-one cases collected by me—that the loop of small intestine places itself immediately above the narrow mesenterial pedicle of the sigmoid flexure, just as we described its doing in twisting of the intestine. Through the space which is now left between the posterior abdominal wall and the pedicles of the two loops crossing each other, one of them, the sigmoid flexure, passes, after having crossed again in front of the loop of small intestine, going from below upwards (Fig. 15, diagrammatic). The small intestine forms the axis about which the sigmoid flexure is wound.

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[1] The same process sometimes follows the development of a large round tumor in the mesentery. Eppinger's interesting case in the Prager Viertelj. 1873, 1, is an example.

[2] For fine examples of this, I refer to *Fagge*, l. c., S. 352. *Rokitansky*, l. c., and *Froriep*, Chir. Kupfert. 370. VIII. 78. *Ruehle* and *Busch* (meeting of the Niederrhein. Gesel. in Bonn, 15th March, 1865). *Eppinger's* finely described cases, l. c.; also, *Moritz's* (meeting of the allg. Ver. Petersb. Aerzte. 5. March, 1866); the cases in the Berichten aus. d. k. k. allg. Krankenh. in Wien, 1865, S. 214; 1866, S. 131; 1858, S. 89; 1862, S. 76; 1868, S. 157; and many others.

[3] *Eppinger*, l. c.

[4] *W. Gruber*, Virch. Arch. Bd. 26 and 48. *Küttner*, ibid. Bd. 43. *Herberg*, ibid. Bd. 54.

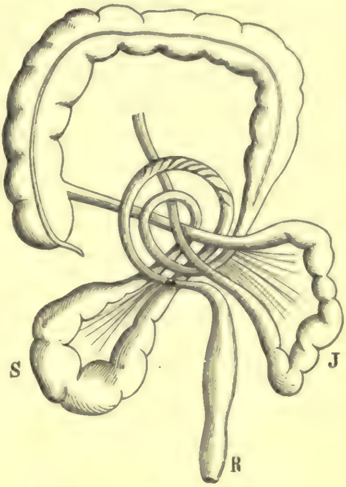

FIG. 15.

It more rarely happens that, after the loop of the ileum has fallen across the mesenterial pedicle of the sigmoid flexure, the former is wound about the latter, and not the latter about the former, as in the case just described. The sigmoid flexure then constitutes the axis. The knot can also be made by the ileum passing with its mesentery under the sigmoid flexure, and then either the ileum or the flexure is wrapped around the other. In this way we may distinguish four kinds of intestinal knots, according as the sigmoid flexure or the ileum forms the centre, and according as the ileum passes at first over or under the pedicle of the sigmoid flexure. In all closely studied cases of knotting, the length of the small intestine proved to be unusually great, and, as a rule, considerably above the average. Almost always in the cases described, the loop of small intestine, as well as the sigmoid flexure, was twisted about its own mesenterial axis, and, as usually happens, more or less torsion of the intestine about its own longitudinal axis was added.

In all the cases within my knowledge, with one exception (Eppinger), knots of this kind have been formed only in males, and always in adults between the ages of twenty-four and seventy-three years. *The course is extraordinarily acute*, as rapid as in strangulation due to any other cause. In one case (Parker) death occurred on the sixth day, in all the others within the first two days, and in many of them on the day of the attack itself.[1]

The length, which is generally considerable, of the strangulated loop,[2] and the tightness with which the knot is usually drawn, lead to the more or less complete occlusion of numerous large mesenteric veins, infiltration of blood, and gangrene of the strangulated intestinal walls, while free hemorrhage into the bowels, and abundant sero-sanguinolent exudations into the peritoneal cavity, often take place very promptly. The latter, for example, in a case of Küttner's which lasted only twenty-four hours, amounted to six pounds.

If the necessary anatomical conditions exist, a traumatism of the abdomen, an error of diet, or vigorous peristaltic action set up by any other cause, may suffice, as numerous examples prove, to produce the formation of the knot. Severe diarrhœa, especially, is apt to precede this kind of internal strangulation, and in some rapid cases it lasts through the whole of the attack, and is associated with free vomiting, complete collapse, etc., a train of symptoms similar to that of cholera.

Knotting or intertwining, exactly like that we have been considering, takes place in rare cases between two suitable loops of the small intestine—that is, loops with long, stretched mesenteries; they cross each other, and while one forms the axis, the other winds itself about it, passing between the root of the mesentery and the loops. I saw an exactly similar occurrence several years ago. A loop of the small intestine was adherent at *a* (Fig. 16), near a hernial opening. About this, as an axis, the adjoining ileum, *b*, had wrapped itself, and sunk into the true pelvis, where it lay as a bluish red, half gangrenous mass, completing the strangulation by its own weight. Cases of a similar kind have been communicated by Rokitansky (l. c.) and Eames.[3]

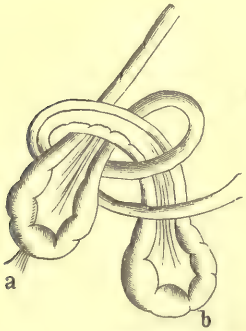

FIG. 16.

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[1] For example, a case lasting only twelve hours, recorded in the Bericht a. d. k. k. allk. Krankenh. Wien, 1866, S. 145.

[2] The loop of small intestine forming the knot varies in length between four and twenty-one inches, that of the sigmoid flexure between twelve and forty inches.

[3] Med. Press and Circular, 1868. Published records, so far as I know, contain only six cases of knots of the small intestine. The many descriptions of knots formed by

Finally, we have still to mention that knotting and intertwining, such as we have described in the foregoing, may, in rare cases, take place also between a loop of the jejunoileum and the cæcum and ascending colon, rendered freely movable by an unusually long mesentery. The latter is displaced inwards, sometimes as far as the left hypochondrium, and at the same time receives a twist about its mesenterial axis. About the mesenterial pedicle thus formed, a portion of the jejunum, rendered freely movable by a long mesentery, winds itself,[1] or vice versa—that is, the ascending colon, twisted about its axis and displaced towards the median line, wraps itself about the small mesenterial pedicle of a long loop of the jejunoileum.[2]

Twisting about the mesenterial axis should be distinguished from twisting about the axis of the intestine. The latter occurs as *real twisting* only at the ends of an intestinal loop twisted about its mesenterial axis (page 567). *What has otherwise been also included in this term should much more properly be designated as lateral kinking* (*the result of dislocation*)[3]. This process is seen much the most frequently in the cæcum and ascending colon. When these are rendered movable by an unusually long mesocolon and displaced inwards or into the left hypochondrium, they suffer at the same time a twisting, or rather a kinking, about the longitudinal axis of the intestine. The angle of the kink may lie near the cæcum, or higher up on the colon ascendens,[4] and always runs more or less obliquely to the axis of the intestine. The kink may, however, also lie in the transverse axis of the intestine ; this happens in the following way : the abnormally movable cæcum, extending into the pelvis and free from its mesentery to an unusual extent, is thrown from below upwards upon the anterior surface of the ascending colon.[5] *But*

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two intestinal loops, as they are offered to us even now, are so far from exact, and often so confused, that the lack of a clear conception of the anatomico-pathological relations is at once evident from the description. A large part of the statistics is equally useless, since the anatomico-pathological account is limited to the statement that a "volvulus of the colon or ileum" existed, and the general term, volvulus, includes all possible causes of internal strangulation, even invagination.

[1] Ber. a. d. k. k. allg. Krankh. in Wien, 1858, S. 331, 332.

[2] *Leonard*, Arch. gén. de méd. T. V. 4, p. 242.

[3] In contra-distinction from the drawing or kinking by adhesions and traction, described before, we are now speaking of the kinking that results from displacement.

[4] Examples given by Habershon, Fagge, Rokitansky, Lieutaud, Cruveilhier, Froriep, and many others.

[5] Examples given by *Rokitansky*, l. c.; *Weiglein*, Oesterr. med. Jahrb. V. 1833 ; *Moutard-Martin*, L'Union, 1856.

the kinking is, in itself, insufficient to produce definitive impermeability, which requires the addition of some compressing cause, most frequently the mesentery of a convolution of the small intestine which overlies the bent point and obstructs it by compression. Under these circumstances, the cæcum always appears as an enormously distended pouch, and often fills the entire mesogastrium. Kinking or twisting about the intestinal axis has been seen not only in the cæcum and ascending colon,[1] but also in the duodenum (Remboldt,[2] Lebert, Poland),[3] and the lowest part of the ileum, the latter resulting from lateral dislocation of the cæcum and ascending colon, or of the ileum itself.[4]

The kinking of the ascending colon and cæcum, described in the foregoing, may, like the twisting of the sigmoid flexure about its mesenterial axis, take on the chronic form, in which case peritonitic adhesions make the angle permanent. Sometimes no noteworthy obstruction of the permeability of the intestine results, as I was recently able to convince myself upon a cadaver; in other cases the symptoms of stenosis appear. Acute occlusion follows in such cases, either from compression of the angle, or from obstruction of the fæces with hyperextension and paralysis of the displaced cæcum.

We have repeatedly spoken of compression of the sigmoid flexure, of the cæcum and ascending colon[5] displaced inwards, by the mesentery of the small intestine; we shall now only add that the lowest part of the ileum, fastened down by a short mesentery, may be closed in the same way. Compression of the intestine by the mesentery sometimes persists, as I have repeatedly convinced myself, as a chronic condition, and gives rise, for a long time, only to the signs of habitual constipation. In most cases of compression of the intestine by the mesentery, diarrhœa preceded the symptoms of strangulation, and it is of importance, etiologically, to this extent, that probably it is often the cause of the overloading of the compressing convolution of the ileum, and of its taking its place upon the compressed intestine. The course is usually very acute. This kind of occlusion occurs almost exclusively in adults, most frequently in advanced life, more rarely in women than in men.

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[1] Paterna (Dissert. Inaug. Berlin, 1872) describes also such a one of the transverse colon in the neighborhood of the splenic flexure. The case was seen in Frerichs' Clinic.

[2] Oesterr. Ztschr. f. pract. Heilk. 1856. 6.

[3] Guy's Hosp. Rep. 1843.

[4] *Guéneau de Mussy*, Gaz. hebd. 1867.

[5] *Erichsen*, Petersb. med. Zeitschr. 1862.

Under the title, "compression of the intestine by other portions of the same," are described many cases of intestinal occlusion, which, in part, stand in great need of another explanation. Thus, Rokitansky found the flexura hepatica occluded by the pressure of the ileum displaced into the right hypochondrium. Others report that the sigmoid flexure, unusually distended and filled with fæces, has sunk into the pelvis and occluded the rectum by pressure; others, again, that the ileum, or even the transverse colon, when disposed in the form of the letter M, has been compressed by an unusually distended sigmoid flexure.[1] In most of these cases, so many of which have been recorded, the difficulty has lain in the unusual size and abnormal course of the colon, which, disposed in many folds, often takes up so much space in the abdominal cavity as to leave scarcely any for the small intestine crowded to one side.[2] In these cases, as a rule, severe coprostasis precedes the occlusion, and this latter is much less the result of compression of the intestine by itself than it is of an insurmountable coprostasis associated with paralysis and hyperextension of the intestine.

The abnormal arrangement of the colon in numerous curves and flexures is, as we have before said, often congenital.

Thus Jacobi found the following condition of things in a child, that died when only five days old, with symptoms of strangulation. The transverse colon went directly down from the flexura hepatica to the left iliac fossa, and there formed several Ω-shaped loops lying upon one another, whose numerous bends hindered the advance of the fæces, and by reciprocal pressure and paralysis brought about at last their permanent arrest.

In other cases the abnormal size and arrangement of the colon is acquired; it is due to habitual coprostasis, and especially is often the consequence of eating excessive amounts of coarse vegetables or of indigestible substances. Consequently it is often found in gluttonous idiots. We shall recur to this subject when we come to speak of occlusion of the intestine by fæcal obstruction, by hyperextension and paralysis of the intestine ("ileus paralyticus").

After compression of the intestine by the intestine and its mesentery, let us consider the other causes of occlusion by compression. We distinguish *acute compression* caused by the sudden change of place of a movable tumor (for example, the lodging of a pedunculated ovarian cyst, of a fibroid of the uterus in the pelvis), or by the engagement of a movable loop of intestine between an immovable tumor and its solid base. We also distinguish the far more frequent chronic compression, usually preceded by adhesion between the compressed portion of the intestine and the body which exerts the compression. In the latter case the symptoms of constriction of the intestine are usually present for a long time before occlusion occurs. The latter may take place in different ways: by a rapid increase or a change of place of the compressing cause, by obstruction of the fæces with paralysis and hyperextension, or by the lodging of a foreign body at the point narrowed by the compression and adhesion. It is evident that the parts most frequently compressed must be those which are fixed by a short mesentery or an incomplete peritoneal investment to the posterior abdominal or pelvic wall, so that they cannot escape: thus the rectum, especially, the lowest portions of the ileum, and the duodenum.[1] As a result of the local conditions and other forces, compression occurs most frequently—in seventy-one per cent. of the cases—in the true pelvis. No further explanation is needed of the fact that this kind of impermeability happens more frequently in females than in males.

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[1] *Buchanan*, Froriep's Notizen. XV. No. 17. *Easton*, quoted by Duchaussoy, l. c. *Oulmont*, Bull. de la soc. anat. 1842, p. 336. *Höhne*, Dissert. Vratislav. 1841.

[2] *Mosler*, l. c. S. 114.

For examples of compressing causes, I refer to the numerous cases of compression of the rectum or other portions of the intestine by uterine and ovarian tumors, by retroflexion and retroversion of the uterus, especially when it is enlarged by pregnancy (Meigs[2], Putegnat[3]), by abscesses and tumors having their origin in the walls of the pelvis, the sacro-iliac synchondrosis, etc.; also compression by pessaries,

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[1] My statistics of 165 cases of compression (exclusive of compression by the intestine and its mesentery) give:

Compression	of the	rectum in	60	per cent.
“	“	lower part of ileum	10	“
“	“	sigmoid flexure, colon descendens and flexura sinistra together	12	“
“	“	duodenum	7	“
“	“	ascending colon and flexura dextra	6	“
“	“	middle portion of ileum	4	“
“	“	the transverse colon	1	“

[2] Quoted by Duchaussoy, p. 372.

[3] Journ. de méd. de Bruxelles, 1867.

prostatic tumors, large vesical calculi (Tulpius[1]), by tubal pregnancy (Hipp. Boscus[2]); compression by subperitoneal tumors and abscesses, or those having their origin in the vertebral column, by abscesses by congestion, psoas abscesses, perityphlitic phlegmons; compression by various tumors of the omentum, of the mesentery, by cysts of the latter (Ball,[3] Sutherland[4]), by cysts of the fossa ileocœcalis (Schott,[5] Widerhöfer[6]), by echinococcus cysts (Lesauvage,[7] Hauston[8]), acute compression by the pedicle of a movable kidney (Rollet[9]), of a movable spleen (Alonzo,[10] Baimbrigge,[11] Helm, and Klob[12]), by tumors arising in the liver or spleen (Blasius[13]), by kidney stones, hydronephrosis, and other tumors of the kidney; compression of the duodenum by tumors arising from the concavity of the liver, from the portal lymphatic glands, by cancers, abscesses, cysts of the pancreas (Kerkringius,[14] Holscher,[15] Bleuland,[16] Habershon (l. c.), and others); compression by the engaging of a portion of the intestine between the ribs and the convexity of the liver (Lavater,[17] Kellenberg[18]), etc.

Obstruction of the Intestinal Canal by Gall-stones, Intestinal Stones, Foreign Bodies, and Fæcal Masses.

Obstruction by Gall-stones.

It is known that even large gall-stones can be evacuated per anum without giving rise to any trouble or annoyance. In other cases, their passage down the intestine is accompanied by severe colics, irregular evacuations, meteorism, and vomiting. In a third set of cases, the gall-stone is arrested at some point in the intestine, and there blocks up its lumen. This occlusion may be permanent, and cause death. It is often overcome, sometimes after the most severe symptoms of internal strangulation have appeared accompanied by stercoraceous vomiting lasting for several days (Wilson, Marotte, and others). The passage is sometimes freed suddenly with immediate remission of the severe symptoms ; this happens when the gall-stone obstructs the lower part of the ileum, and passes suddenly into the cæcum, or when it is evacuated after having lodged in the rectum. It is not always upon the size of the stone that the one or the other of these occurrences depends. The shape also is of importance. Thus a long cylindrical stone with a small transverse diameter may pass without causing any trouble, while a much smaller one, if it is spherical and has a greater diameter, may occlude the intestine. The size of the intestine also, which varies within physiological limits, as well as accidental causes, have some influence.

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[1] Observ. Lib. III. C. 2.

[2] De Facult. Anat. Lib. 2, p. 23.

[3] Bull. de la soc. anat. de Paris. 1857, p. 400.

[4] The Medical Record of Australia, 1863.

[5] Wochenbl. d. Zeitschr. d. k. k. Gesell. d. Aerzte in Wien, 1862, No. 44.

[6] Jahrb. d. Kinderh. II. Jahrg.

[7] Bull. de la facult. de méd. à Paris. 1813.

[8] Catalogue of the Mus. Coll. of Surg. in Ireland. No.

[9] Path. u. Ther. d. bewegl. Niere. Erl. 1866.

[10] Arch. gén. de méd. 4. Série. T. XVI. p. 506.

[11] Ibid. p. 505.

[12] Wien. Wochenbl. 1856, 37.

[13] Quoted by Duchaussoy.

[14] Obs. anat. 43.

[15] Schmidt's Jahrb. III. Suppl. Bd. 168.

[16] Mus. anat. No. 1053.

[17] Thes. Inaug. 5. C. l. c.

[18] Diss. Inaug. Erl. 1861.

It is a fact, proved by many observations, that even large gall-stones can pass gradually through the distensible bile ducts, and in this way reach the intestine. Abercrombie (l. c., S. 166) found in a fatal case of lodgment of a gall-stone in the small intestine (the stone measured four inches in longitudinal and three and a half in transverse circumference) the ductus choledochus communis so enlarged that it would admit a finger.[1] In other not infrequent cases a fistula forms between the gall-bladder and the duodenum, and the stone makes its way through it into the intestine. It is exceedingly rare that the expulsion takes place into the colon through a fistula between it and the gall-bladder.[2]

The gall-stone may lodge at any point in the small intestine, most rarely in the middle of the ileum, more frequently in the duodenum and jejunum, most frequently—and here I go against other opinions—in the lowest part of the ileum, one or two inches above the ileocæcal valve.[3] The cause lies in the diminution which the calibre of the intestine undergoes as it approaches the cæcum, and partly also in the fixation of the lower part of the ileum by a short mesentery.

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[1] Compare *Morgagni*, Ep. XXXVII. Art. 46.

[2] *Murchison*, Lond. Path. Soc. Vol. XXII.

[3] Of the thirty-two cases of obstruction by gall-stones in which the position of the stone is given, contained in my statistics, it occupied the duodenum and jejunum in 10
The middle of the ileum in.. 5
The lowest part of the ileum in.. 17
32

Sometimes it happens that after the gall-stone has made its way into the lowest part of the ileum, it remains there for a long time, obstructing it only incompletely, and gives rise to a set of symptoms composed of those of constriction of the intestine, and repeated temporary occlusions. (Meteorism, colics, visibleness of the hypertrophied folds of the intestine: attacks which exactly resemble hepatic colic, except that they are associated with much meteorism.) Circumscribed peritonitis results from the irritation excited by the gall-stone in the intestinal walls, and produces annular narrowing of the ileum at the corresponding point, which finally reaches such a degree that the engaged gall-stone becomes fixed and causes death with the symptoms of permanent occlusion. Sometimes the stone, by remaining in the canal for a long time, pushes out its wall on one side in the form of a false diverticle. Such diverticles have been found in the duodenum (Chomel,[1] Harly [2]) as well as in the ileum (Habershon, l. c., S. 263). Finally, the rarest termination is that of evacuation of the gall-stone through the abdominal wall with formation of an intestinal fistula. A stone remaining for a long time in the canal or in a diverticle may grow by the superposition of fresh layers by the passing contents of the intestine. In this way are formed stones having a radiating cholesterine nucleus in the centre, and a shell composed of the phosphate of ammonia and magnesia, and the phosphate of lime, in which, however, may be mingled decomposed bilirubin and a small amount of cholesterine. A gall-stone, that has been lying in a diverticle, may, as a result of vigorous peristaltic action, or because it grows more and more into the lumen of the canal, make its way out of it into the intestine, and either occlude the latter or be evacuated per anum.

Fatal obstruction by gall-stones has been observed only in the small intestine. Still the most severe, though temporary, symptoms of impermeability of the colon have been repeatedly caused by the lodging of one or more choleliths in the sigmoid flexure or rectum.[3] When there is stenosis of the ileum or colon, occlusion can be caused by smaller stones. As for sex, of the forty-one cases of my statistics, thirty-two were females, nine males.

As the relative frequency of gall-stones in men and women was, according to Hein's excellent statistics, as two to three, it is perhaps proper to infer that large gall-stones capable of causing occlusion are formed more frequently in women. As for age, it happens almost always in late life, after the age of fifty, and indeed by far the most frequently after the age of sixty years; in only a few exceptional cases was the age less than fifty.[1] Since gall-stones are not correspondingly more frequent in later than in early life, we must conclude that by a long stay in the intestine they gradually increase to a size which is sufficient to cause occlusion.

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[1] Hist. de l'acad. roy. 1710. Obst. anat. 3.

[2] Trans. of the Path. Soc. of London. Vol. VIII. 235.

[3] *Leo*, quoted by Henoch. l. c. *Ehrlich*, Gaz. med. de Paris, 1845, 167.

A few days ago Prof. Liebermeister showed in his Clinic a gall-stone which had completely occluded the cæcum not far from the small intestine. The patient, a man a little over sixty years of age, had died with symptoms of ileus lasting for seven days. The concretion now lying before me is one of the largest of those found in the fifty-two cases of fatal obstruction by gall-stones and also of those that have been passed per anum. It has the form of the gall-bladder, and in the centre is composed of a radiating nucleus of cholesterine shining like silk. The other layers are made of a mixture of bile pigment, chalk, and cholesterine. As it has been proved chemically and microscopically that the latter is present in large quantities, this is a real gall-stone. Its length is seven and a half centimetres, its greatest circumference, that according to which it was lodged in the intestine, is 12.7 ctm., its greatest diameter four ctm., its weight 51.2 grammes.[2]

Ordinarily, intestinal and gall-stones can be distinguished from one another by their simple appearance. The light weight of most large gall-stones, their radiated structure, their glistening appearance, and the fatty feeling of the cholesterine stones, is very characteristic. In doubtful cases, the distinction can be made by chemical analysis. For the clinical diagnosis, it is sufficient to treat a small piece of the stone in a test tube with alcohol, and then with nitric acid. The question is decided by the well-known rings of color formed by the bile pigment about the fragment of the stone, which, being of light specific gravity, usually floats in the liquid. The altered bile pigment of the intestinal stone does not give the same result. Another fragment may be boiled in alcohol, and then suddenly cooled. The cholesterine separates in glistening flakes, often in the form of a thick milky magma, which, on being shaken, shows the silvery glister of the cholesterine scales, and it can also be easily recognized by means of chemical and microscopical examination.

The symptoms of obstruction by gall-stones vary according to the location of the same. If this is in the duodenum, vomiting begins at once, and furnishes, until death, abundant quantities of bile. The collapse is rapid, the abdomen retracted, the urine scanty or lacking. The course is peracute, and death may ensue in from six hours (Büchner, l. c.) to two days. When the obstruction is situated in the lower part of the ileum, notable meteorism, limited at the beginning rather to the hypo- and mesogastrium, occurs, and the vomiting becomes fæcaloid and then feculent. The secretion of urine is not always diminished. The duration of the disease in these cases varies from four to seven days. Lodgment of a gall-stone sometimes follows immediately after a severe attack of hepatic colic, in other cases a long time, three years, for example, in Murchison's case, after such an attack, or after the symptoms of peritonitis in the region of the liver; which latter led to adhesions between the gall-bladder and duodenum, and thus prepared the way for the breaking through of the stone, a process which, as Habershon justly remarks, may, however, remain entirely latent. In chronic cases of incomplete obstruction, definitive occlusion is preceded for a long time by the symptoms of diminished permeability of the small intestine, and sometimes by the repeated but temporary appearance of the severe symptoms of total obstruction with ileus. The diagnosis of obstruction by gall-stones can, under favorable circumstances, be made with probable accuracy.

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[1] The youngest person with obstruction by gall-stone is one mentioned by Peacock: twenty-seven years old. Lond. Path. Soc. Vol. I. 255.

[2] Almost as large, or even somewhat larger, gall-stones occluding the intestine are mentioned by *Büchner* (Henle and Pfeuffer's Zeitschrift. XI. 1850), 8 ctm. long, 3½ wide, weight 30 grammes. *Pye Smith*, Lond. Path. Soc. Vol. V. 163. 13 ctm. in circumference. *Van der Byl*, ibidem. Vol. III. 231. 11½ ctm in circumference, 4½ ctm. in diameter. *Baly*, Lond. Path. Soc. Vol. X. p. 184.

Obstruction by Intestinal Stones.[1]

Stony concretions formed in the intestinal canal—*enteroliths*—occur far more frequently in certain ruminants and solipeds (under the names of bezoars and ægagropilæ[2]) than in human beings. In the latter they are formed and situated mainly in the colon, especially in the cæcum[3] (typhlolithiasis, Albers), or in deep sacculi of the colon, or in false diverticles, or in the rectal ampulla. In rare cases enteroliths have been also found in the small intestine, not far from the ileocæcal valve, or in its diverticle.[1]

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[1] *Marcet*, on the chemic. hist. and med. treatment of calculous disorders, 1817. *M. Laugier*, Mém. sur les concretions, Paris, 1825. *Forbet* and *Duncan*, Edinb. Med. Journ. Vol. XXIV. *A. Monro*, Morb. anat. 25. *Jäger*, Ueb. d. Darmsteine des Menschen und der Thiere. Würzb. 1834. *Copland*, Encycl. Wörterb. II. 3. 341. *Albers*. Arch. f. physiol. Heilk. 1851. *Meckel*, Arch. f. d. Phys. Bd. I. S. 454. *Jules Cloquet*, Gaz. de Paris. 5. 1855, and in the Bull. de l'acad. des sciences. 1850. *Lehmann*, Lehrb. d. phys. Chemie. 2. Aufl. Leipzig. 1853. II. 124. *Virchow*, in Virch. Arch. Bd. 20. 1860.

[2] From αἲξ ἄγριος, chamois, and πῖλος, hair, known as "hairballs" (Gemskugeln). The word *bezoar* is said to be derived from the Arabic, *badesar*, antidote (?).

[3] Cases given by Monro, l. c.

The older literature is rich in communications on the subject of enteroliths. But very often the description of the appearance of such stones, their weight, etc., leaves no doubt but that they were gall-stones (choleliths), a remark which Monro justly made about Morgagni.[2] By the test used in ancient times, based upon the theory that gall-stones would float and intestinal stones sink in water, many gall-stones would pass for enteroliths.

If we restrict the term intestinal stone, not applying it to every unusually hard scybalon, I think, on looking over the literature of the subject, that the following principal varieties should be considered as real enteroliths:

1. Heavy, stony concretions, showing concentric layers when divided, often with a chalk-like, dirty-white centre, and, towards the periphery, with alternating light and dark, coffee or chocolate-colored layers, of which the one on the outside is usually the darkest. This disposition also occurs in the reverse order, from the periphery towards the centre.[3] The concretions are composed in great part of phosphate of lime; also of phosphate of magnesia and of ammonia and magnesia, organic substances and water.[4] Sometimes the centre contains more lime, the periphery more magnesia. Other stones are composed mainly of phosphate of magnesia and of ammonia and magnesia, and the latter is sometimes found upon the surface in large fine crystals (Virchow, l. c.). Stones of this kind are formed especially in the cæcum, the vermiform appendix, a diverticle of the colon, in rare cases, also, of the ileum, and in large pouch-like sacculi. The concretions increase in size very slowly, as is proved by the length of time during which they are often known to exist, and seldom become larger than a chestnut.[5] Sometimes there are

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[1] Thus in a case observed by Weber, and described by Albers (in the text of the Path. Anat. Atlas), a real enterolith passed from the ileum into a pocket of the ileocæcal valve and caused total obstruction. An enterolith of the ileum (ileolith) is mentioned by Monro, l. c., p. 66.

[2] Ep. XXXVII. Art. 24.

[3] See the plates in the ordinary anatomico-path. atlases. Also *Madelung.* Arch. f. klin. Medic. V. 122. *Monro*, l. c., Plate II.

[4] Aberle (Würtlemb. Corresp.-Bl. XXXVIII. 1868), for example, found the following percentages: water, 22; phosphate of lime, 60.5; phosphate of magnesia, 4.3; sulphate of lime, 1.1; extractive matter (alcohol and ether), 0.3; other organic constituents, 11.3.

[5] Still, larger ones have been known: one 11 ctm. (Simson), another 19 ctm. (Dyer), and a third, 23 ctm. (Monro) in circumference, and one of 23 ounces (Simon), and another of 4 pounds (Monro d. Ae.) in weight.

several such stones together. In the case observed in Niemeyer's Clinic thirty-two stones were evacuated, weighing together two and a half pounds; and in a case of Monro's 12. (See also Klebs l. c., S. 288.) The form is generally rounded, oval, more rarely with facets when there are several together. They often have a foreign body at the centre, especially a fruit-pit (Aberle, Simson [1]), a piece of bone (Monro), a gall-stone (Portal), or indigestible pieces of vegetables (*e. g.*, licorice fibers, Laugier), sometimes also various other things, as a nail, eggs of the ascaris, (Klebs), a splinter of granite (Simon [2]), etc. It seems to me worthy of remark, in opposition to the opinion generally held, that stones of this kind are almost always seen in young people or those of middle age.

2. Enteroliths of low specific gravity, generally of irregular, ramified form, and of the consistency of compressed sponge. They have fine pores, and when examined with a glass, show a network of fibres, the interstices of which contain indurated fæces and earthy or solid chalky substances. These concretions are sometimes perforated wholly (Ballonius), or in part (Monro), or enclose several large cavities. They are composed of a felted mass of indigestible vegetable fragments. They are formed principally in the cæcum, from whence they may make their way to different points in the colon. Their size is ordinarily between that of a chestnut and that of an orange, and there are seldom more than two together.[3]

Concretions of this kind are by far the most frequently described as "oat-stones" (Avenoliths), composed of the closely felted fibres and husks, and the caryopses of panicled plants, with a large amount of earthy phosphates. Concretions of this kind, according to the testimony of many physicians, have been observed very frequently in Scotland, where the poorer people eat much coarsely prepared oaten grits and bread. But other suitable vegetable fibres and husks sometimes form intestinal stones of this kind when closely felted together and impregnated with lime salts (Albers, l. c.) Stones of this kind are also observed in most cases in young people or those of middle age.

3. Stones, whose formation is due to the long-continued use of certain mineral medicines. Bamberger [4] observed an intestinal stone containing eighty per cent. of the carbonate of lime in a patient who for years had eaten freely of chalk. Pure magnesia stones have been formed in the same way (Cloquet, l. c., Brande,[5] Henry,[6]

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[1] Observ. de la soc. méd. d'Edinbourg. T. I. 363.

[2] *Buchner's* Repert. XVI. 215. See also *Porta*, Arch. gén. 1. Sér. T. XII. p. 432.

[3] Monro speaks of a stone weighing six ounces, and measuring ten ctms. in length, nine in breadth. Huss and Mosander (Mus. anat. Holmiense, fasc. I. figs. 1 and 2. Holm. 1855) speak of one seventeen ctm. long, six ctm. wide. Turner (Gaz. méd. de Paris. 1843) tells of fourteen concretions, one of which was round and had a diameter of seven ctm. Three years afterwards, eighteen other concretions came away. (See *Harley*, Lond. Path. Soc. XI. 87.)

[4] Krank. d. chylopoët. Syst. S. 423.

[5] Quoted by Cruveilhier, l. c.

[6] Quoted by Monro, l. c., p. 34.

and others), also an enterolith composed of magnesia and iron, and measuring thirty-seven ctm. in circumference (Hutchinson[1]), and a benzoar stone (Prof. Erichsen[2]), etc. Stones of this kind also are formed chiefly in the cæcum.

Only in very rare cases do enteroliths occlude the intestine suddenly without having given rise to premonitory symptoms. This happens only with those stones which are formed in diverticles of the small intestine, and then suddenly make their way into the intestine and occlude it. In most cases grave trouble in the abdomen, leading to emaciation and hypochondria, and sometimes all the signs of diminished permeability of the intestine precede this for a long time,[3] or frequently repeated attacks of typhlitis and perityphlitis, until finally death is caused by perforation or complete obstruction of the intestine, or complete recovery follows the evacuation of the stone (Monro, Albers, and many others). In these cases a tumor can often be felt in the abdomen which, taken in connection with the cachectic appearance of the patient, is not unfrequently supposed to be cancer, until it disappears and leaves no trace after evacuation of the concretion.

Obstruction by Foreign Bodies.[4]

Foreign bodies, swallowed in consequence of carelessness, fright, mistake, or with a view to suicide (most frequently by children, lunatics, hysterical individuals, and jugglers), are often evacuated per anum after a longer or shorter period, and after having caused more or less trouble, sometimes none at all. This has happened even when the object was quite large.

The most various kinds of objects have gotten into the intestines in this way and been evacuated again, partly without having caused any trouble; thus, for example, large pieces of bone, glass-balls, stones, needles, coins, in a few cases even large pieces of wood and iron, keys, knives, a fork, dagger-blade, small flute, a spoon, whetstone, etc. Schröder van der Kolk saw a lunatic who evacuated per anum on the third day after he had swallowed them, a door-key eleven ctm. long, and a nail twelve ctm. long. A. Rothmund[1] extracted from the rectum a piece of iron eight and a half Paris inches long, and as thick as the little finger, on the nineteenth day after it had been introduced by the mouth. Annandale[2] saw a child three years old pass per anum a breastpin five ctm. long, which it had swallowed a short time before. The same author relates that London counterfeiters, when arrested while passing their money, used to swallow it, and afterwards procure its evacuation by eating large quantities of cheese and hard-boiled eggs. In many districts the ingestion of a large amount of raw cabbage is a popular means employed with a similar object. In Zoja's[3] experiments upon dogs and cats, needles were evacuated in from twenty-four to thirty-six hours after having been swallowed. Not unfrequently, as examples have shown, large foreign bodies remain for a long time, even for years, in the stomach until they cause death by perforation, or escape in consequence of the formation of a gastro-colon fistula, or per vias naturales. We constantly find cases recorded in which balls of hair[4] (cheveux enchevêtrés) have been formed in the stomachs of women, who, as a result of hysteria, lunacy, or a bad habit, have made a practice for years of swallowing hairs, wool, or yarn. These balls grow to a considerable size, manifest themselves by a tumor in the epigastrium, and cause death either by the complete arrest of digestion with chronic vomiting and signs of inanition, or by perforation, or because the mass of hair has made its way into the duodenum, or ileum, and occluded it. In one of these cases the accumulation of the hair took place in the cæcum, ultimately obstructed the ileocæcal valve, and caused death by ileus (Teft[5]).

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[1] Transact. Lond. Path. Soc. VI. p. 203.

[2] Ibid. XI. 86, et seq.

[3] In Watson's case (Edinb. Med. Journ. XII. 1868), thirty years, in one of Monro's five, in another seven years, in Turner's four, in Simson's six years. In a case recorded by Paterson (Edinb. Med. Journ. 1868) severe attacks of ileus occurred repeatedly.

[4] *Peter*, Arch. gén. de méd. Sept. 1855. *Cloquet.* ibid. T. V. 369. See also *Günther*, Lehre v. d. blut. Operat. l. c.

A foreign body that has passed from the stomach into the intestines seldom causes immediate and acute occlusion. Ordinarily it tarries for a long time in the duodenum, lower ileum, cæcum, or rectum,[6] and there excites the more or less severe symptoms of partially obstructed permeability. The escape of the foreign body through the abdominal walls—this happens more rarely into the vagina, or from the ileum into the colon or rectum—occurs most frequently from the cæcum, the favorite resting-place of foreign bodies.

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[1] For the literature of foreign bodies, S. A. aus d. "Deutschen Klinik," 1859, No. 15, et seq.

[2] Edinb. Med. Journ. 1863. VIII. Other interesting cases are mentioned by Cruveilhier, l. c. *Winge*, Schmidt's Jahrb. Bd. 92. S. 114. *Pilcher*, Lancet. 1866. *Bamberger*, l. c., S. 428.

[3] Virch.-Hirsch's Jahresb. 1867 and 1868.

[4] Examples may be found in: *Cruveilhier*, Mém. de l'acad. roy. de méd. 1777. *Pollok* (Lond. Path. Soc. Vol. III. p. 327), a mass of hair fourteen inches long, two inches thick. *G. May* (Assoc. Journ. 1855). *Best* (Brit. Med. Journ. 1869), a mass of hair weighing thirty ounces. *Russel*, Times 1869. *Inman*, ibidem.

[5] Schmidt's Jahrb. Bd. 135. S. 75.

[6] Not unfrequently also in portions of the intestine which constitute hernias (*J. L. Petit*, *Plunque*, *Critchett*, Lond. Path. Soc. I. 268, and many others).

We include here, also, obstruction of the intestine by indigestible masses which have been taken with the food. Usually these are accumulations of fruit-pits, grape-skins, husks, fibres, and other vegetable remains, which, without the aid of a previously existing stenosis, produce an obstruction (by accumulation) of the colon or rectum, more rarely of the lowest part of the ileum.[1]

The appearance of peritonitis often helps essentially in such cases to make the impermeability permanent.[2] Not a few of the recorded cases of "recovery from ileus" were obstructions of this kind by accumulated, indigested masses, which, after the gravest symptoms had appeared, were removed spontaneously, or by suitable means. Foreign bodies and large indigestible masses play an important part when the intestine is constricted. They either collect above such a constriction and ultimately occlude it by their bulk,[3] or a single fruit-pit, a potato-peel, the endocarp of an apple, a piece of tendon, a bone, or even a hard bit of fæces, is sufficient, if lodged in the stricture, to cause permanent occlusion. Foreign bodies give rise more frequently than gall or intestinal stones, to a constriction by cicatricial bands, or chronic peritonitis, at the spot where they have remained for a long time. Thus foreign bodies that have been swallowed, and afterwards evacuated per anum, may be significant for the diagnosis of subsequent constriction of the intestine.

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[1] *Marshall*, Med. Chir. Trans. XXXV. p. 65, mentions an obstruction-by-accumulation of the duodenum by a pound of needles that had been swallowed.

[2] Examples of such obstructions-by-accumulation without stenosis: *Laugier*, Mém. de l'acad. de méd. T. I. p. 413, 1828. *Quain*, Lond. Path. Soc. V. 145 (four pounds of cocoanut fibres). *Langdon-Down*, ibid. XIII. 1867, also cocoanut fibres. Furthermore *Dewastine* (quoted by Duchaussoy), obstruction by seven hundred cherry-stones. *Donovan*, Gaz. méd. de Paris, 1848. *Margérie* (L'Union, 1867). *Lafon*, Virchow-Hirsch's Jahresber. 1869. *Makintosh*, Arch. gén. I. XXVIII. 408.

[3] Thus *Cruveilhier* saw six hundred and seventeen cherry-stones collected above a stenosis, *Molard* a mass of fig-seeds, *Compton* currant-seeds; similar cases mentioned by *Louis*, *Marchesseau*, *Caron*, *Bristowe*, *Legg*, *Kaltschmidt*, *Schröder v. der Kolk*, *Bourdon*, and many others.

More frequently than by the mouth, foreign bodies make their way into the rectum by accident, through malicious design, in children irritated by worms, in lunatics and idiots, through unnatural gratification of the sexual appetite, and they often remain there for a long time, in the capacious ampulla, without causing any annoyance.

In other cases, especially when they get higher up, they cause more or less complete obstruction, or kill by perforation, peritonitis, ulceration of the cellular tissue of the pelvis, etc. The records contain abundant examples of the most singular objects which have gotten into the intestines in this way : bottles, glasses, pomade-pots, forks, wooden pegs, tallow-candles, a pestle, coffee-cup, iron pincers, a pigtail, etc. For details we refer to the surgical text-books.[1]

At a time when it was the fashion to consider intestinal worms responsible for every possible disease and symptom of disease, we cannot be surprised to find recorded numerous examples of an ileus verminosus, supposed to be due to occlusion of the intestinal canal by masses of worms. After a careful examination of the literature bearing upon this point, I must consider the occurrence of fatal obstruction of the intestines by masses of worms as not proven,[2] without, however, wishing to deny that they might aid in producing definitive occlusion when constriction was already present. In many cases of fatal illness with vomiting, colics, and constipation, which remained unexplained by an incomplete autopsy, the earlier physicians were contented to consider some mass of worms that was discovered as the cause (for example, in peritonitis, perforation, etc.).

Occlusion of the Intestinal Canal by Masses of Fæces. "Ileus Paralyticus."

Conditional upon Habitual Constipation.

We have in mind here those occlusions of the canal which are due simply and solely to the insufficiency of the forces destined to move the contents of the intestines forward, and whose ultimate paralysis leads to permanent arrest of the advance of the fæces and to the symptoms of ileus. We have here to deal mainly with chronic conditions, which, without the coexistence of constriction of the canal, have yet been associated for a long time with the symptoms of habitual constipation, have even sometimes caused temporary occlusion, and have, finally, led to permanent occlusion, under certain conditions yet to be considered. Many of the "cures of ileus" by electricity, massage, mercurius vivus, tobacco, belladonna, etc., which are so frequently met with in medical literature, are nothing but severe coprostases which have gone on to fæcaloid vomiting, and have yielded spontaneously (increase of the catarrh, exudation into the canal, and softening of the obstructed masses of fæces), or to injections, and have terminated in recovery with the evacuation of an often considerable quantity of fæces.[1]

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[1] See *Günther*, Lehre v. d. blutigen Operation, l. c. *F. Esmarch*, in Pitha-Billroth's Hbd. d. Chir. III. Bd. 2. Abth. 5. Lief.

[2] *Cox's* cases also (Edinb. Med. Journ. 1859), *Neuffer's*, Württemb. Corresp.-Bl. 1861, and still less the much-quoted one of *Parkinson* (Schmidt's Jahrb. 1837. XIII. 40), in my judgment are not demonstrative.

To produce ileus paralyticus, it is sufficient that a long portion of the intestine should be incapable of peristaltic action. The intestinal contents collect in this, and oppose an obstacle to the peristaltic action of the portion of intestine lying above it, which is greater as the paralyzed piece is longer. A further obstacle to the advance of the contents arises from the fact that the intestine lying below the paralyzed portion—as is always the case below occlusions—becomes contracted (contraction of inanition), and thereby an increased opposition is made to the advance of the contents which have become consolidated into a uniform mass. Another consequence of the paralysis is the hyperextension of the paralyzed portion; the accompanying stretching and separation of the circular muscular fibres destroy the last chance of a return of the peristaltic power. Finally, the peritoneum of the paralyzed portion usually becomes sooner or later the seat of more or less inflammation, which leads to the serous infiltration of the muscular layer, and renders it incapable of contraction. Not unfrequently, also, does peritonitis play a more primary etiological part; for, starting, for example, from a stercoral ulcer of the cæcum due to chronic coprostasis, it may give rise to paralysis and hyperextension of the corresponding portion of intestine and to permanent arrest of the fæces ("ileus inflammatorius" of the ancients).

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[1] Cases of this kind in *Lavergne's* Clin. de Montpellier, 1843.

Usually, permanent occlusion, produced in the above-described way by the complete abolition of the forces which provide for the advance of the fæces and defecation, is preceded by prolonged and ultimately severe coprostases. Still, paralysis of a portion of the intestine plays an important part under various other circumstances in the definitive arrest of the fæcal current. An existing obstacle, for example, whether it be stricture, bending, or compression, can for a long time be compensated for by hypertrophy of the muscular coat. Finally, this compensation abates, the muscle relaxes, stagnation follows with hyperextension, paralysis of the intestine above the obstacle, and permanent arrest of the movement of the fæces. Circumscribed peritonitis above the occluded point is not necessary in this case to cause the paralysis, although it often does so. It is still simpler when the intestinal wall is infiltrated by a cancer which, without causing any considerable constriction, makes a portion of the intestine incapable of peristaltic action, and in this way stops the advance of the fæces. A loop of intestine, that has been strangulated in an external hernia and fortunately reduced, may have been so completely paralyzed, partly by the peritonitis and partly by the injury inflicted by the strangulation and taxis, that the symptoms of strangulation continue until death. Furthermore, those rare cases in which, during diarrhœa (Annesley[1]), or tuberculosis of the intestine (Haug, l. c.), or typhoid fever,[2] or in the course of a severe chronic intestinal catarrh (Mosler[3]), death follows with stercoral vomiting and the other symptoms of impermeability of the intestine, while no mechanical obstruction can be found at the autopsy, cannot be explained otherwise than by serous infiltration, degeneration, and relaxing of the muscular coat, especially in the neighborhood of large typhoid, tuberculous, or dysenteric ulcers, leading to a paralytic condition of the muscular coat, and thereby to arrest of the advance of the contents of the intestine.

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[1] Researches on the diseases of India, Vol. II. See also *Griesinger*, Ges. Abhdlg. II. S. 686; *Mercier*, Gaz. méd. de Paris, 1867.

[2] *Bamberger*, l. c., S. 160.

[3] Arch. d. Heilkunde, 1864, V. Jahrg. S. 113.

Acute diffuse peritonitis produces functional weakness of the muscular coat as a result of its serous infiltration. Continuous constipation and meteorism result. (The first is usually lacking only in cases of puerperal peritonitis.) Since now, too, the other symptoms of acute peritonitis are sometimes completely covered by those of acute internal strangulation, and especially as stercoraceous vomiting may be absent in the latter when the course is rapid, it may become impossible in many cases to say whether we have to deal with an acute internal strangulation or with a very acute (foudroyante) peritonitis excited by some unknown cause.

Less rarely do we find recorded cases in which, in the course of a partial or general peritonitis (this has been observed most frequently in circumscribed peritonitis about the cæcum, in typhlitis[1]), the functional weakness of the muscular coat due to serous infiltration has increased to total functional incapacity; even stercoraceous vomiting followed meteorism and constipation, and completed the symptoms of internal strangulation. It is evident that in such cases of ileus paralyticus the diagnosis may be incorrect. Bauer (this Cyclopædia, Dis. of the Peritoneum) mentions a case of tubercular peritonitis, with stercoraceous vomiting, in which not a sign of mechanical obstruction could be found at the autopsy. I myself saw, a short time ago, a quite similar case of chronic tubercular peritonitis, which ended fatally, in consequence of an acute exacerbation of the peritonitis, causing constipation, which lasted fourteen days, and stercoraceous vomiting. The autopsy showed the calibre of the canal normal at every point. There was recent, together with older, peritonitis. The meteoristic loops of the small intestine were adherent to one another in many places, and were so united by innumerable old adhesions into a uniform conglomeration, that the enterotome was passed with difficulty through the labyrinth of the convolutions. So long as the contractility of the intestine was not lost in this case, the contents could be moved forward normally, notwithstanding the numerous attachments. But when the acute exacerbation of the peritonitis caused paralysis of the muscular coat, this was no longer possible, and the signs of impermeability appeared.

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[1] *Bamberger*, l. c., S. 160. *Tillaux*, Bull. gén. de thérap. 1870; also the interesting case in Petersb. med. Ztg. 1860. XIV. S. 374.

Finally, we include also in ileus paralyticus those rare cases, coming, however, from trustworthy sources,[1] in which a severe injury to the abdomen—*e. g.*, a kick by a horse—has been followed by all the symptoms of occlusion of the intestine, and yet no mechanical obstruction was found at the autopsy. Here, too, we must seek the cause in paralysis of the injured portion of the intestine, although in many of these cases it remains doubtful whether the traumatism has produced the paralysis and the fæcal vomiting directly or by the intervention of the peritonitis which it has excited.

In rare cases complete paralysis and death, with symptoms of ileus, have been observed in affections of the spinal cord. An interesting case of this kind, in progressive ascending degeneration of the spinal cord, is recorded in the Berichten des k. k. allg. Krankenhauses in Wien (1865. S. 69).

Before we discuss the conditions under which accumulation of fæces and habitual constipation lead to permanent occlusion of the intestine, I must give a brief consideration to the causes of habitual constipation.

If the healthy adult has, upon an average, one or two stools within the twenty-four hours, containing from one hundred and twenty to one hundred and eighty grammes of fæces and seventy-five per cent. of water, there are yet frequent variations lying within the limits of health, both as regards the number of the stools and the quantity of the fæces, which depend upon numerous forces not always clearly understood. The easiest for us is the insight into the causes which produce the normal variations in the quantity evacuated. The investigations of Bischoff and Voit[2] have shown that this depends far more upon the quality—abundant with vegetable, small with animal diet—than upon the quantity of the food ingested, so long as this does not exceed the maximum of digestion. Less clear are the causes of the physiological variations in the number of daily stools in different men. There are healthy adults whose bowels move only every two or even every three days;[1] while, on the other hand, others enjoying perfect health defecate three times daily. In both cases the quantities evacuated are about the same, if the periods compared are long and the subsistence the same. But, as a longer sojourn of the fæces in the colon increases their consistency, we are not surprised to find variations of from fifty to seventy-five per cent. in their watery constituents. If we seek the cause of these differences in the periodicity of the evacuations, which are unaccompanied by any disturbance of health, we find that they depend almost exclusively upon purely individual variations from the average normal activity of the peristaltic action. In such cases of habitually infrequent defecation there is as little morbid atony of the intestine as there is of "atony" of the heart in those who have only forty-five pulsations in the minute.

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[1] *Hildreth*, Philad. Med. Rep. 1871. *Petrenz*, l. c. *Gouzée*, Canstatt's Jahresber. 1843. III. S. 425.

[2] D. Ges. d. Ernährung d. Fleischfresser. Leipzig and Heidelberg, 1860, S. 289.

As for pathological constipation, it is a symptom of different pathological processes and of very varying significance. While, as a temporary condition or as the accompaniment of various affections, it has scarcely any real clinical interest, yet, in other cases, in constriction and occlusion of the intestine, it belongs among the symptoms which are most important in the diagnosis and prognosis. In a third set of cases constipation has a certain independence; it causes a series of symptoms in different and distant organs, and is the starting-point and the most prominent symptom of an affection which the practising physician meets with constantly, and for which, considering the uniformity of the symptoms composing it and the differences in the individual cases in its very often uncertain etiology, the symptomatic title of "habitual constipation" is justly maintained.

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[1] *Habershon*, on diseases of the abdomen, Lond. 1862. p. 441. *Henoch* (l. c. S 479) tells of a woman, sixty years old, who from youth upwards had had a passage from the bowels only every six or eight days, and whose health had been perfect. Such cases must depend, not exclusively upon functional, but, as in similar published cases, also upon anatomical variations from the normal, especially upon a congenital abnormal length and arrangement of the convolutions of the colon.

Excluding now those cases in which the constipation is due to a mechanical obstruction, to a constriction of the intestinal canal, the ultimate cause of most of the other kinds lies in a sluggishness or weakness of the peristaltic action. The causes of this are extraordinarily manifold. Not unfrequently many causative forces are at work at the same time, and it often cannot be determined which was the primary one or the most important.

Let us now consider somewhat more closely some of the causes of constipation.

Increased excretion of water through the skin and lungs or the kidneys causes constipation, unless the greater loss is compensated for by abundant drinking. Here it is partly due to a more rapid absorption of the liquids taken into the alimentary canal, and partly to a reduction of the proportion of water, and also of the quantity of the digestive juices poured into the intestine. The consequence of this quickened absorption and diminished secretion is a thickening of the intestinal contents, occurring not only more promptly, even in the ileum, but also going on to a greater degree especially in the colon. And now, whether it is that drier masses, especially in the ileum, offer more opposition to normal peristaltic action, or because the contents of the intestine, in consequence of increased absorption of water and diminished secretion of the intestinal juices, lose those stimulating qualities which enable them at other times to excite reflex peristaltic action, the evacuations become less frequent and firmer, and constipation appears. On the other hand, free drinking of water, if the amount that can be absorbed is less than that taken in, often causes diarrhœa, and the relation between absorption by the intestines and excretion of water through the skin, lungs, and kidneys, which is entirely analogous to what has been already mentioned, shows itself in the reduction of the diuresis, of the perspiratio insensibilis, the dryness of the tongue, the thirst when the evacuations are profuse, and also in the appearance, which is sometimes noticed, of profuse diarrhœa when the function of the kidneys is suddenly arrested. But just as under these circumstances the diarrhœa depends upon a contemporaneous increase of the peristaltic action, so in the cases of constipation due to an abnormally increased loss of water, the cause must be thought to lie mainly in a consequent diminution of the peristaltic action. The constipation which appears during increased perspiration, under different physiological and voluntarily established conditions (sweat cures), or during certain diseases (acute rheumatism, phthisis, intermittent fever), the constipation of puerperal women (increased by the relaxation of the abdominal pressure which favors the emptying of the rectum, and by the loss of water caused by the secretion of milk), and the constipation which accompanies diabetes, all must receive the same explanation. The constipation which is often seen in fever is due, partly to the increased escape of water through the skin and lungs, partly to the reduced secretion of intestinal juices caused directly by the fever, and partly to the less amount of nourishment taken. The constipation observed during "fasting cures" depends upon the two last-named causes, the constipation of voluntary abstention from water (Schroth's cure[1]) upon a reduction of the amount of water drunk, and the constipation accompanying stricture of the pylorus upon a reduction in the amount of water and nourishment taken, and upon a consequently diminished secretion of intestinal juices.

If the normal admixture of digestible food with the digestive juices furnishes the physiological stimulus which, in a reflex manner, excites peristaltic action, so can qualitative, like the quantitative, changes in this stimulus give rise to anomalies in the activity of the intestine, to diarrhœa or constipation. The abnormal quality of the intestinal contents depends either upon the ingesta or the digestive juices, or both. As for the ingesta, we are acquainted with many medicines and articles of food which excite diarrhœa or constipation. According as the action of these articles is temporary or chronic, so do they excite temporary or chronic anomalies in the evacuations. On the other hand, an injury operating for only a short time, may, by exciting permanent anatomical changes—acute or chronic catarrh—disturb for a long time the functions of the intestine. We find great individual differences in the reflex excitability of the intestinal canal. Ingesta, which cause abnormal stimulation of the intestine and diarrhœa in one person, have no effect upon another, and each individual, at different times, is affected in different ways by the same intestinal stimulant.

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[1] *Juergensen*, Deutsches Arch. f. klin. Med. I. S. 217. See the tables.

As for constipation caused by the ingesta, we have already spoken of that which is due to the taking of an insufficient amount of water into the alimentary canal: thus in stricture of the pylorus, in individuals who neglect to moisten the masticated food suitably, or who make use of nourishment, which, when judged by the amount of liquid taken, is too dry. We have also mentioned the constipation due to insufficient nourishment or to entire deprivation of nourishment. Also, the prolonged use of food that furnishes but little residue, *e.g.*, milk diet, sometimes causes not only scanty but also infrequent evacuations. The same effect is produced by great uniformity in the kind of food taken every day, for the sensibility of the canal is thereby diminished. While, for example, the milk diet not unfrequently produces diarrhœa in those who are not accustomed to it, yet when its exclusive use is continued uninterruptedly, unfrequency of the stools is observed. In individuals whose diet for a long time has been of this unstimulating kind, the use of coarse bread increases the peristaltic action. On the other hand, the habitual use of coarse vegetable food, of much bread and potatoes, induces habitual constipation, partly because an abatement, a sort of fatigue of the peristaltic force, ensues as the result of the increased activity which the intestine has to show in order to dispose of the large residuum, just as purgatives employed for a long time finally have the opposite effect.

Besides articles of food, we have many medicines which cause constipation. Our knowledge of the exact mode of operation of most of them is exceedingly incomplete.

Mucilaginous substances, mucus, and gums, have a constipating effect, probably because they accompany the ingesta throughout the canal, envelop them, and diminish their stimulating influence upon the mucous membrane. The way in which the astringents (alum, tannin, red wine, and the numerous vegetable astringents) retard peristaltic action, whether by an astringent effect upon the mucous membrane of the intestine with reduction of its reflex excitability, or by diminishing the secretion of the intestinal juices, and causing the contents of the intestine to thicken promptly, is still very uncertain. Many of these remedies—tannin, for example—have proved to be entirely without influence upon the healthy intestine, and in larger doses have caused acute catarrh and diarrhœa. Some believe, therefore, that the astringents have a constipating effect only upon a mucous membrane that is catarrhally inflamed. This view is opposed by the simple experience that astringent red wines always cause constipation in those who are not accustomed to them. As to the immediate cause of the constipating effect of nitrate of silver, bismuth, chalk, etc., we have nothing but hypotheses. We know but little more of the cause of the constipation in chronic lead poisoning, for the theory that it is due to cramp of the intestine caused by irritation of the inhibitory intestinal nerves, the splanchnic, is only a circumlocution for known facts. Opium is thought to act only by diminishing the reflex excitability. But just as the constipating effect of lead is thought by many to lie in a capacity to excite spasm directly through the motor intestinal ganglia, so, too, it cannot be denied that opium has a direct paralyzing influence upon that apparatus.

As for the constipation caused by diminution in the amount of the digestive juices, we have already alluded, when speaking of that due to free withdrawal of water through the skin or kidneys, to that occurring during fever, and that caused by a reduction in the amount of water or nourishment introduced into the body. In the jaundiced, if the passage of the bile into the intestine is hindered, constipation is a regular symptom. It has thence been inferred that bile is a stimulant of peristaltic action. I am more inclined to ask if the retarded peristaltic action is not rather the result of accumulation of the biliary acids in the blood, which may affect the movements of the intestines as they do those of the heart, especially as the nervous system of the intestine is very similar to that of the heart (ganglion cells, inhibitory and exciting fibres). In chronic cases of icterus due to obstruction, the chronic catarrh certainly influences the reduction of the peristaltic action. Moreover, the fact that in icterus due to obstruction, the not inconsiderable quantity of liquid which, under normal conditions, flows every twenty-four hours into the intestine with the bile, is withdrawn, may, under certain circumstances, assist in producing constipation. The way in which other anomalies in the quantity and quality of the digestive juices secreted aid in checking peristaltic action is unknown.

Peristaltic action of the intestine is not only excited by reflex irritations acting upon its mucous membrane, but the centres of innervation, lying in the brain and spinal cord, also send directly exciting and inhibiting impulses to it through the pneumogastric and splanchnic nerves. Furthermore, the intestine possesses "independent" centres of innervation lying within its walls, in the form of a double ganglionary system, of a plexus myentericus Auerbachii, situated between the longitudinal and circular muscular layers, and of a nerve plexus in the submucosa discovered by Meissner.

Just as joy and fear, or great fright, sometimes excite such active peristaltic action that diarrhœa ensues, so do we find, also, that conditions of mental depression (hypochondria, melancholia) and sometimes hysteria also (tympanites hystericus), notably reduce the activity of the intestine. From these cases, in which habitual constipation is the result of a hypochondria arising from central causes by a purely mental process, must be distinguished those in which, on the contrary, the habitual constipation becomes the starting-point of a mental derangement which may go on to the grave forms of hypochondria.[1] To the interruption of motor nerve trunks must be attributed those cases of constipation which are always observed in certain affections of the spinal cord associated with paralysis of the limbs (myelitis, degenerative processes, transverse separations), and in many of these cases the consequent paralysis of the abdominal muscles, which aid materially in emptying the rectum, must also be taken into consideration.

Constipation due to a condition of tonic contraction of the intestines in meningitis (especially acute hydrocephalus) and other acute cerebral affections, cannot be attributed to paralysis of motor nerves, but rather to irritation of inhibitory ones. Here, too, the accompanying condition of tonic contraction of the abdominal muscles (as is most prominently the case in tetanus) seems to share with other factors (abundant perspiration, frequent vomiting) in causing the constipation.

For many, bodily exercise is a means of exciting peristaltic action. (Its influence is often paralyzed by profuse perspiration.) The more frequent respiration thereby induced, the increased action of the diaphragm and abdominal muscles, seem to act like massage of the abdomen, a procedure which was employed in ancient times to promote action of the bowels. It is possible, also, that the change in the distribution of the blood, caused by bodily activity, has some influence, for every change in the supply of blood to the intestine seems to excite peristaltic action (Schiff, Donders). On the other hand, constipation affects the bedridden and those who lead a very sedentary life. In many cases the lack of bodily exercise causes constipation, indirectly, by means of a chronic intestinal catarrh. In other cases this relation between constipation and intestinal catarrh is reversed.

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[1] *Griesinger*, Path. u. Therap. d. psych. Krankh. 3 Aufl. S. 221, 236, and 201.

Finally, we have a series of constipations whose cause lies in changes in the muscular coat itself of the intestine. We have already spoken of constipation accompanying peritonitis, and have traced it to the fact that the muscular coat of the intestine shares in the inflammatory processes of the adjoining peritoneum by undergoing serous infiltration and a consequent disturbance of function. Perhaps the constipation observed in Bright's disease depends upon a similar process, upon œdema of the intestinal walls, including the muscular tunic. The constipation which appears during convalescence from severe febrile affections, and which yields as the nourishment of the patient improves, has its cause most probably in the fact that the intestinal muscles also share in the parenchymatous degeneration set up by the fever in all the other muscles. The constipation occurring in the course of many chronic diseases and cachexiæ accompanied by emaciation, and that which comes with old age, depend upon defective nutrition, degeneration, diminution, and functional weakness of the muscular coat. Atrophy of the intestine shows itself in anæmia, thinness, and softness of the intestinal loops. In many of the last-named cases of constipation, other factors have shared in its production, such as too little food, profuse perspiration, bodily inactivity, deficient secretion of digestive juices, chronic catarrh.

Chronic intestinal catarrh is one of the most frequent causes of habitual constipation.

While diarrhœa usually accompanies acute catarrh, in consequence of increased excitability of the intestinal mucous membrane (it is generally so, too, in cases of intestinal ulcer), the chronic catarrh of adults is always associated with constipation. It sometimes extends over the entire canal (when the portal circulation is obstructed, whether directly by cirrhosis, tumors occupying the concavity of the liver, or indirectly by affections of the heart and lungs which have led to inefficiency of the right ventricle and to obstruction of the flow through the vena cava), sometimes over only a large portion of the ileum or colon. The cause of constipation in chronic catarrh lies in the diminution of reflex excitability due to swelling, tumefaction, and hypertrophy of the mucous membrane. Perhaps, too, it is aided by changes (which have been made too much of in many quarters) in the quality and quantity of the digestive juices—the secretion of a very tenacious mucus, which acts like a solution of gum. The principal cause of the constipation is to be found unquestionably in the muscular coat itself, which in all protracted cases participates in the abnormal nutritive processes carried on within the mucous membrane and becomes flaccid, in the same way as do the bronchial muscles in chronic bronchitis, the muscular coat of the bladder in chronic cystitis, and that of the stomach in gastric catarrh. Another important result of chronic catarrh is the diminution of the elasticity of the intestinal walls. As a result of this, and the relaxation of the muscular coat, the production of meteorism is favored. Under healthy conditions, every great accumulation of gas within the intestine increases the intra-intestinal pressure; the increased pressure excites peristaltic action, perhaps by pressing upon the intestinal ganglia, just as considerable augmentation of pressure in the aortic system quickens the action of the heart; and in this way every great accumulation of gas in the healthy intestine is at once relieved. In chronic catarrh, on the other hand, the diminished elasticity of the walls prevents the accumulation of gas from increasing the intra-intestinal pressure; peristaltic action, therefore, is not excited, and the condition becomes chronic.

Chronic intestinal catarrh is one of the most frequent causes of habitual constipation; in this etiological category, especially, belong a great number of the cases of habitual constipation with hypochondria, which the practising physician encounters so frequently. From the history of such patients we learn that their trouble has usually lasted a long time, and that it gradually developed insidiously and progressively with repeated remissions and exacerbations. A close examination often makes it plain that the mental depression appeared later, and is a secondary symptom. We learn less that is definite about the real cause of the disease than about its beginning, for the most of what the patients have to say with reference to the etiology bears the mark of morbid reflection upon their own condition. The patients usually are men between twenty and forty years of age. Certain occupations, especially sedentary ones (such as shoemakers, weavers, booksellers, school-teachers, clerks), are frequently represented among them. The complaints and troubles of the patients are most various. A sense of oppression and obstruction of respiration, palpitation of the heart, and a feeling of pressure or fulness in the abdomen, are seldom lacking, symptoms which are due to the existing meteorism and the elevation of the diaphragm caused by it. In addition, they often complain of faintness, ringing in the ears, rush of blood to the head, headache, abnormal sensations of heat and cold in the extremities, pains in the sacrum and back, dragging and dull pains in the genitals, and ischiatic pains, or pains in the adductor muscles. We are far from being able to give a sufficient causative explanation of even a small part of these abnormal sensations and neuralgias.

The favorite theory, that these pains are caused by the pressure of the overloaded intestines upon branches of the lumbar plexus (nerv. genito-cruralis, obturatorius, cutaneus femoris externus) or the plexus ischiaticus, is certainly applicable to only a limited number of cases ; with still less propriety are they referred to venous hyperæmia of the meninges of the spinal cord, to spinal irritation, and the like. Chronic gastric catarrh is almost always present at the same time, and it can by itself, without chronic intestinal catarrh, give rise to the same set of symptoms of habitual constipation with hypochondria. I have repeatedly met with cases of habitual constipation in hypochondriacs in which examination of the patient revealed the signs of ectasia of the stomach—that is, of a palpable lesion as the cause of the whole condition. The accompanying chronic gastric catarrh causes other troublesome symptoms: pressure, and fulness, temporary pains in the region of the stomach, hypochondria, eructations, perverted taste, pyrosis, and loss of appetite. The digestive disturbances which are associated with chronic catarrh of the stomach and intestine, affect the general nutrition; the patients after a while become pale and emaciated with an unhealthy complexion and physiognomy.

As in other chronic affections of the abdomen, which produce rather a persistent feeling of discomfort than real localizable pain, so the morbid irritations in habitual constipation conducted by the sympathetic nerves to the brain, excite in the patient an indefinite obscure feeling of obstruction, an injury to his Ego, and cause uneasiness, which becomes chronic, since the abnormal symptoms proceeding from the abdominal organs, the feeling of diffused pressure, of dull weight, and fulness, continually force themselves upon the attention and demand explanation. The inability to obtain this explanation increases the mental uneasiness, and soon excites the foreboding and fear of a threatening serious illness. If afterwards abnormal sensations of more definite meaning appear here or there—often they are only such as would pass unobserved in health—or if actual pains are felt, they are at once noticed, even when they are so slight, by the consciousness lying, as it were, in wait, and their meaning, which is usually negative, is interpreted unfavorably. The friends say that the patient "exaggerates" his pain; and, with the mistaken idea that this is done intentionally and deliberately by the patient, they fail to give him a proper amount of sympathy. The originally sensitive and irritable patient becomes depressed, morose, and loses pleasure in the exercise of his calling; all his thoughts are directed to his physical condition, a marked, constant feeling of illness destroys all enjoyment of life, and robs him of interest in everything that does not relate to his health. While many of these patients give their whole attention to the functions of the intestines, and conscientiously record every evacuation, its color, quantity, and other peculiarities, note the frequency of the calls, the escape of wind, and the bleeding of hemorrhoids, as something important, and make every presumably unusual appearance the source of fresh anxiety and an excuse for another consultation with the physician, others are led by abnormal sensations in distant parts to consider the actual cause of their suffering, the habitual constipation induced by chronic catarrh, as simply a concomitant symptom of another affection. It so happens that after such patients have recounted all their physical ailments, it is often necessary to ask a special question, in order to learn that the movements of the bowels are habitually delayed. On the other hand, they talk of pains in the sacrum and back as the supposed signs of a grave affection of the spinal cord; a feeling of oppression and palpitations of the heart are supposed to point to disease of the heart or lungs, dryness and soreness in the throat to the beginning of "tracheal consumption," a tawny pale-yellow color of the skin to a serious affection of the liver, abnormal sensations in the genitals (perhaps due to pressure of the overloaded intestines upon the spermatic veins, the genito-crural nerves, and the plexus pudendalis) to impotence, sexual exhaustion, and impending disease of the spinal cord. Misled by reading popular medical books, the patient believes he has every disease of which he can find a single symptom (even the most unimportant) in himself. He is often reminded by this reading of sins of his youth, committed perhaps many years before, and these then serve to explain all the symptoms from which he is suffering. Statements made by patients of this kind concerning spermatorrhœa, frequent involuntary emissions by day, while at stool or urinating, depend usually upon gross delusions, even if it is not to be denied that occasionally difficult defecation may by pressure upon the seminal vesicles cause involuntary evacuation of their contents. Such patients not unfrequently show in their descriptions the influence of popular medical literature, and enable us to see clearly that many of their morbid anxieties have their origin in those dirty writings which, under the dissembling title, "the medical counsellor," and composed with no other object than that of pecuniary profit, speculate upon the curious and hypochondriacal: upon the first, because they promise to reveal to the uninitiated, in a spicy manner, the "secrets of sexual life;" and upon the latter, because they lay all human pathology to the account of onanism and youthful excesses, the results of which they paint in the most exaggerated colors. It is known that patients of this kind are animated by a lively desire to recover, but that they seldom hold fast to any directions and follow them out. Usually they wander from one physician to another, and, as they are considered to be hypochondriacs, they often receive neither proper attention nor requisite sympathy, and so fall into the hands of dealers in secret remedies, homœopaths, and "nature doctors," subject themselves in turn to the Swedish-movement cure, to all kinds of bathing, air, and drinking cures, and often spend large sums of money upon the swarms of secret remedies advertised in the newspapers. A cure is often found by such patients only after they have followed the strict *régime* of a suitable watering-place, or have made use of an accurately ordered dietetic and curative as well as attentive mental treatment in a rationally conducted institution. I have seen obstinate, well-developed cases of habitual constipation with hypochondria permanently cured in this way, together with the use of cold baths for many weeks, and by taking aloes pills every evening for several months.

Habitual constipation resulting from chronic intestinal catarrh depends, as we have already explained, upon deficient activity of the muscular coat, and is situated mainly in the small intestine. It is evident that an abnormal fæcaloid thickening of the contents of the small intestine must result if their progress is retarded. This can be proved by the constipation caused by affections of the heart or liver with development of chronic intestinal catarrh. It is also evident that if the contents of the small intestine contain but little water when they pass into the colon, they must become unusually solid, even if they remain in it for only the normal length of time. Furthermore, if the contents of the ileum pass infrequently into the colon, infrequency of the stools, constipation, results, even supposing the colon to possess its normal activity. In fact, examination of the patient shows that probably in many of these cases of habitual constipation caused by chronic intestinal catarrh, the colon is not involved. Under such circumstances we find the meteorism limited to the meso- and hypogastrium (meteorism of the small intestine), with relative depression of the parts corresponding to the colon, and, on the other hand, repeated examination never shows fæcal tumors in the latter. This proves that the activity of the colon is normal, and that the constipation depends upon the fact that the contents of the small intestine are passed infrequently into the colon on account of the diminished peristaltic action. The colon is very often in a condition of great contraction (inanition-contraction) under these circumstances of primary constipation in the small intestine, just as it is below a stenosis. Its peristaltic action is then retarded, for a certain moderate distention of the intestine with gaseous and other matters is necessary to its normal activity. As this distention increases, so, too, does the peristaltic action ; if it falls below a certain degree, the other becomes quieter. The contracted condition of the colon is shown by the fact that the stools are formed not of continuous masses but of single, small, round or many-sided scybala which can often be felt on palpation arranged like a rosary in the descending colon and sigmoid flexure. Of the same kind, and in fact for the same reasons, are the evacuations in cases of stenosis situated high up in the colon or in the ileum, and often, too, in ectasia of the stomach (see p. 517) and under circumstances of starvation ("starvation intestine").

In contrast to the habitual constipation which has its origin in the small intestine comes that which arises especially or primarily in the colon, and which we designate by coprostasis in the narrow sense of the word. It is plain that an abnormal weakening of the peristaltic action of the colon, united perhaps with insufficiency of the abdominal pressure which is essential to the emptying of the rectum, causes constipation even when the peristaltic action of the small intestine is normal. Sometimes the coprostasis is even accompanied by increased peristaltic action of the small intestine, for in some cases of fæcal stagnation (evidenced by constipation and by fæcal tumors that can be felt in the descending colon and sigmoid flexure) abundant moist borborygmi of the small intestine can be heard, if the ear is laid upon the abdomen, and the activity of the peristaltic action is thereby demonstrated.

As for coprostasis, or constipation arising in the colon, a detailed consideration of its causes would render many repetitions unavoidable, for many of the causes of constipation already described affect the colon as well as the small intestine: for example, many cases of constipation due to chronic catarrh, abnormal excretion of water by the skin or kidneys, atrophy and atony of the intestine in old age or in chronic cachexia.

Nevertheless, we must make special mention of coprostasis: first, because many of the circumstances belonging to it have a certain independence in their causes, symptoms, and course; and, secondly, because permanent, fatal, fæcal obstruction may, although rarely, be caused by them.

We have now to speak of the *abnormal length and arrangement of the colon*, an anomaly the lowest degree of which so often presents itself as the M-shaped depression of the transverse colon into the hypogastrium, while its higher grades are associated with the formation of many abnormal flexures and S-shaped curves. This abnormal arrangement of the colon was often mentioned by the older writers (such as Ruysch, DeHaën, and others) as something important, described with great detail, and not unfrequently illustrated by many sketches. In Esquirol's time far too much weight in the etiology of mental diseases was attributed to this and to the coprostasis which was believed to be its necessary consequence—going so far as to establish the so-called copro-psychiatria;[1] and even quite recently the pathological significance of these abnormal flexures of the colon has been extraordinarily exaggerated.[2] This anomalous disposition of the colon, so often met with in those of every age, is in most cases, so long as it does not become excessive, an entirely insignificant symptom, which, as experience teaches, causes no trouble and not even constipation. Normal peristaltic action compensates as adequately for the obstacles offered by the more numerous curves as it does for those of the normal flexures. But if a notable diminution of the forces which advance the fæces and empty the rectum is superadded to the above-described disposition of the colon, coprostasis follows, and is the more severe as the abnormal flexures are more numerous, more angular, and longer.

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[1] See *Griesinger*, Path. u. Therap. d. psych. Krankh. S. 201, et seq.

[2] *Vötsch*, Die Koprostase. Erl. 1874.

The abnormally convoluted arrangement of the colon is due in most cases to abnormal conditions of growth and position during fœtal life or early childhood. Different forces, acting alone or together, may influence this. We shall consider only a few of them, leaving a more detailed examination for another occasion. If the cæcum, which in the fourth and fifth months of fœtal life lies in the right hypochondrium below the liver, does not descend completely into the fossa iliaca dextra ("incomplete descensus"), the inevitable result is that the colon, though of normal length, is thrown into abnormal folds. In new-born children the descensus often is not terminated; and it is well known that for this reason, and still more because the colon grows more rapidly in the fœtus than the abdominal walls do, the sigmoid flexure in new-born children projects into the right iliac fossa. Incomplete descensus is occasionally found in adults also, when the cæcum is situated higher than usual. In consideration of the etiology, those cases also belong here in which, while the cæcum occupies the iliac fossa, this position is rendered possible only by an unusually developed mesocæcum, which not unfrequently shows by its direction backwards and upwards from the cæcum that originally the descensus was incomplete, and that it was afterwards made good by a longer mesocæcum. A cæcum (and colon ascendens) so abnormally movable, is liable to displacement with lateral kinking (the so-called "twisting about the axis of the intestine"), to compression by the mesentery of the ileum, and perhaps also plays a part in the etiology of many ileocæcal invaginations.

The longitudinal muscles which appear in the seventh month, the ligamenta coli or tæniæ, shorten the colon, for they draw it together in the direction of its longitudinal axis; they thus produce the tufted appearance of the colon, the sacculi. If the development of the tæniæ is delayed, or if it happens to be too weak, this too contributes to make the colon too long when compared with the abdominal walls. Finally, in many cases of abnormal congenital arrangement of the colon we have to do with primary anomalies in the disposition of the mesentery, the abnormal length of which has resulted in an unusual movability of the colon with formation of anomalous flexures.

The four known factors—incomplete descensus cæci, defective development of the muscular ligamenta coli, abnormal growth of the colon, making it too long for the abdominal cavity, and abnormal length of the mesentery—may act together, and give rise to flexures of the colon, so numerous, and lying upon and beside one another in such a way, that the excretion of the meconium is not only rendered difficult, but, as undoubted observations show,[1] is entirely prevented, a condition which may lead to the death of an otherwise normally developed infant, in consequence of great meteorism and insufficient breathing, or even with the symptoms of ileus.

Besides the factors already mentioned, others exist during intra-uterine life which may give rise to anomalies in the arrangement of the colon and to many unusual flexures. Without going further into the subject—for we should lose ourselves in the details of embryonal development—I shall mention only those cases in which the colon, although of normal length, runs from the normally situated cæcum directly to the left hypochondrium, and thence descends with many flexures to the rectum, and those others in which the colon runs directly from the hepatic flexure to the left iliac fossa, and there forms two or more S-shaped flexures. Finally, we have the abnormal curves which occur when the colon is congenitally placed upon the left side, an anomaly which, as we saw, is usually associated with a common mesentery for the ileum and colon (p. 560).

In other cases the abnormal length and arrangement of the colon is an acquired condition due to habitual coprostasis. In the same way as a colon above a stenosis is often longer and more sinuous, so does the same condition result from chronic coprostasis, for it gives rise to prolonged obstruction, first at one point and then at another.

Furthermore, the loading of different portions of the colon with fæces may cause them to sink downwards and their mesentery to lengthen, just as this not unfrequently happens in (non-adherent) carcinoma of the transverse colon, in which case the tumor can sometimes be felt immediately above the symphysis. The occurrence of abnormal positions of the colon is favored in advanced life especially by the laxness of and loss of fat in the mesentery on the one hand, and the flabbiness of the abdominal walls on the other.

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[1] *Jacobi*, American Journ. 1869. *P. Monterossi*, Deutsch. Arch. f. d. Physiol. von Meckel. VI. Bd. 4. Heft. S. 566, et seq.

This explains why the course of the colon is very often found to be abnormal, and to present many flexures in people who have long suffered from habitual coprostasis. We will study rather more closely some of these cases which are so frequently met with both in practice and in medical literature.

As a result of deficient activity of the forces which empty the rectum (abdominal pressure and contraction of the rectum), habitually incomplete defecation occurs with accumulation of a large amount of fæces in the rectal pouch lying between the lower and the Nélaton sphincters. The hyperextension of this pouch, which often results, increases the flaccidity of the elastic and muscular elements of the rectum. If an individual thus situated becomes bedridden, or otherwise compelled to omit bodily exercise, the intervals between his stools become longer than usual, often five or six days; and now symptoms may appear quite suddenly, which in many respects resemble those of acute occlusion of the intestine. Active peristaltic motions take place, mainly induced by a laxative administered internally, but they are not able to break through or set in motion the hard or tenacious, voluminous masses of fæces accumulated in the rectum or lower part of the colon.

Violent colics set in, together with an eager desire to empty the bowels, which the patient in vain attempts to satisfy with exhausting efforts and straining. Collapse follows, profuse sweat breaks out, the features assume the pinched appearance of the facies abdominalis, the limbs become cold, the pulse small and rapid, the abdomen distended, and not unfrequently vomiting occurs. If the attempt is made to move the bowels by means of injections, the introduction of the rectal sound meets with great resistance, which often makes it impossible to inject any liquid. Nothing remains but to pass the finger into the rectum, and thus remove the hard, clay-like, tenacious fæcal masses. If the first obstacle is thus removed, the way is opened for injections, whose action is greatly aided by diarrhœal masses passed down at the same time from above. The collapse disappears as rapidly as it came, abundant evacuations take place, and in a short time the patient, from a condition of great pain and anxiety, becomes perfectly comfortable.

Cases of this kind are rare in practice; they occur in old people, especially in women whose abdominal walls have become very much relaxed by repeated pregnancies, and in whom the senile atrophy of the pelvic organs and the breadth of the pelvis prevent any considerable obstacle to the hyperextension of the rectal ampulla. A similar condition is also seen occasionally in young people, when the emptying of the rectum is postponed for an unusually long time, on account of severe pain during defecation (inflamed piles, fissure of the anus), or on the employment of abdominal pressure. The records contain numerous examples in which habitual accumulation of fæces in the rectum and sigmoid flexure, especially in old people, has led to permanent arrest of the peristaltic action of the colon, to paralysis with hyperextension, and thus to ileus, which either ended fatally or yielded to proper treatment, with evacuation of enormous masses of fæces.

Habitual coprostasis, due to enfeebled peristaltic action of the colon, is very common in advanced life, in those of sedentary habits, in atrophy of the intestine occurring in the course of chronic cachexiæ, and especially, too, in lunatics. Under all these circumstances, we not unfrequently find, as a result of habitual arrest of the fæces, that the course of the colon is abnormally sinuous. The frequency with which these anomalies are found in the bodies of lunatics led physicians to attribute too much importance to them in the etiology of mental diseases. Habitual or even exclusive nourishment with coarse vegetable food leaving a large residuum, leads not unfrequently, after prolonged use, to abatement of peristaltic power of the colon, to repeated fæcal stasis, to enlargement and abnormal position of the colon. Sometimes extreme degrees of the latter are found in voracious idiots, whose habits of swallowing all sorts of indigestible objects act together with the immoderate use of vegetable food in producing the anomalies presented by the colon.

Thus Little and Callaway[1] report the case of a voracious idiot, thirty-four years old, who died with symptoms of impermeability of the intestines. The transverse colon was six inches in diameter ; the sigmoid flexure extended, as an enormous bag, from the left hypochondrium and left iliac fossa, to the right hypochondrium, filling the entire meso- and hypogastrium. Rampold relates a very similar case.[1]

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[1] Transact. of Lond. Path. Soc. III. 106.

With reference to the difference in the length of the colon, as between those who have been eaters of meat and those whose nourishment has been composed mainly or entirely of vegetables (*e. g.*, the difference between inhabitants of cities and of the country), careful examinations of many bodies, taking into consideration the stature and occupation, promise further results. Thus, the average length in Russia (twenty to twenty-seven feet of small intestine, eight feet of colon) seems to be not inconsiderably greater than in Germany (fifteen to twenty feet of small intestine, six to seven feet of colon). Küttner[2] thinks this is due to the larger amount of vegetable food (in Russia there are one hundred and ninety fast-days in the year !). As a matter of fact, the intestines are longer, in proportion to the length of the body, in herbivorous than in carnivorous animals.

Large fæcal tumors that can be felt are not unfrequently formed under the above-mentioned circumstances of habitual coprostasis. These tumors, as numerous recorded examples show, have often led to a wrong diagnosis, being mistaken for malignant tumors, invagination, tumors of the stomach, omentum, liver, spleen, and kidneys,[3] peritonitic or perityphlitic exudations, ovarian or other pelvic tumors,[4] and even for pregnancy,[5] until the tumor disappeared spontaneously, or under the influence of suitable treatment, with evacuation of large quantities of fæces, and thus revealed its real character. Fæcal tumors differ according to their duration and their size and position. Along the ascending colon they form soft cylindrical tumors, usually not distinctly limited, which feel exactly like invaginations, perityphlitic exudations, or certain tumors of the right kidney. Collections of fæces can be felt most easily in the descending colon and sigmoid flexure, where they give the sensation of a rosary-like row of fæcal balls, between which the colon is tightly contracted (contraction of inanition). It is plain that these contractions, which present as many (functional) stenoses as there are balls of fæces, oppose the progress of the masses. Collections in the transverse colon often lie immediately against the liver, so that, on percussion, it seems to be enlarged. In other cases they resemble tumors of the stomach, omentum, or liver. When the colon is M-shaped, fæcal tumors in the transverse colon may lie immediately above the symphysis. Usually fæcal tumors are painless on palpation, and can be moved when they are situated in movable portions of the colon. Sometimes they feel hard and uneven, sometimes softer; they may be shaped like a sausage, or present large globular masses arranged like the balls of a rosary. It cannot be denied that in certain cases they can be forced along the colon or changed in shape by pressure made through the abdominal walls; but this is so rare an occurrence that, notwithstanding repeated attempts, I have never been able to convince myself positively of it.

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[1] Gaz. méd de Paris. 1847, p. 831.

[2] Virch. Arch. Bd. 43.

[3] An interesting case of this kind is given in the Arch. gén. de méd. T. XX. p. 581.

[4] Thus the celebrated Roux was called to make a four days' journey from Paris, to operate upon a pelvic tumor ; he found a coprostasis of the rectal ampulla, which yielded to the use of injections.

[5] *Laronde*, Presse méd. 1850 ; quoted by *Henoch*, l. c., p. 21. *Frerichs* mentions a similar case, Klin. d. Leberkr. I. S. 73.

It is important to know that fæcal tumors can remain for a long time—even for months—unchanged, at one and the same point, without causing much constipation. Numerous cases are recorded in which old people, who have had normal evacuations daily, and even diarrhœa, have died with symptoms of impermeability of the intestine, and the rectal ampulla and colon have been found filled with hard fæcal masses of astonishing weight. The diarrhœal current, in those cases, found its way between the masses lodged against the walls, often in the sacculi, sometimes also in false diverticles, until by chance some hard lump became engaged in the passage and caused total occlusion. Under certain circumstances, then, habitual coprostasis may last for a long time, and even reach a very great degree, without the patient's being made aware of it by a diminution in the number of stools. Cruveilhier (l. c.) found, in the body of an old woman who died of acute occlusion of the intestine, after having always gone regularly to stool, the rectum and colon filled by an enormous amount of thick fæces, and the colon distended to the size of a horse's colon. In a quite similar case, in an old man, the colon, filled with fæces, measured thirty-five centimetres in circumference, and the cæcum was as large as a child's head. Many similar cases have been mentioned by Bright,[1] Siebert,[2] and many others.

On the other hand, it is not rare to meet, under the above-described conditions, with occlusions lasting from two to four weeks, with accumulation of enormous amounts of fæces and hyperextension of the colon.[3]

We have already seen that prolonged coprostasis, attaining to a notable degree, may, under certain circumstances, lead to paralysis and hyperextension of the colon, and in this way to permanent arrest of the current of fæces and to death, with symptoms of ileus (ileus paralyticus). The same result sometimes follows when, as we have previously said, a paretic, over-loaded loop of the colon, most frequently the sigmoid flexure, sinks into the pelvis, and thereby undergoes kinking, or twisting, or compression by the mesentery of the small intestine; or when the paralytic hyperdistended cæcum is displaced towards the median line, and thus becomes bent or twisted about its longitudinal axis. On making the autopsy of such a case, one is astonished at the size of the colon, or of parts of it, especially the cæcum and sigmoid flexure, which, when seen from in front, overlie everything, and often seem to fill the entire abdominal cavity. Meteorism of the colon, due to paralysis, is often so considerable in such cases that the limited space in the abdominal cavity makes meteorism of the small intestine impossible. The latter, on the contrary, is contracted and pushed backwards, or to one side, by the distended colon.

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[1] Guy's Hosp. Rep. V. p. 302.

Diagnostik d. Krankh. d. Unterl. Erlangen, 1855.

[3] Thus *Chambers* (Digestion and its Derangements, Lond., 1856, p. 496) tells of an extraordinarily large and solid colon in a woman who, while in good health, had a movement of the bowels only once every fortnight. *Levi* (Gaz. méd. 1839) found in a woman, fifty years old, who had had fifteen children, and had suffered for nine years from constipation, the rectal ampulla occluded by a mass of fæces weighing four pounds. *Lemazurier* (Arch. gén. de méd. Sér. I. T. I. 410) found, under similar circumstances, a mass weighing thirteen pounds. *Habershon* (l. c., p. 442) repeatedly found, under such circumstances, the circumference of the colon enlarged to twelve and fifteen inches. Similar accounts are found in *Lieutaud* (Hist. Anat. med. T. I. Obs. 291, 293; *Meckel* (Path. Anat. II. Bd I. Abth. Leipz. 1816, S. 289, et seq.); *Hauston* (Catal. of the Mus. of Surg. of Ireland. Nos. 284, 285, 287); *Trois* (Gaz. des hôp. 1839); *A. Hall* (Monthly Journal, 1846); *Siebert*, l. c.; *Bright*. l. c.; and many others. On the other hand, we have legitimate doubts of cases like the one so often quoted of *Renauldin* (Copland, Encycl. Wörterb. II. 3), in which a woman, fifty-nine years old, was said to have had a movement of the bowels only from four to six times each year, and at her death to have accumulated sixty pounds of fæces in her colon. The same may be said of Mr. Staniland's communication (Medical Gazette, 1832, 1833, p. 246), according to which a man seventy-three years old, who had had only six stools yearly, died after a coprostasis lasting seven months—during which he had had no trouble—from perforation of the enormously dilated cæcum. Mr. Gay tells, in the Pathological Transactions of 1854, of a boy, six years old, who, after a severe fever, was reported to have remained without a movement of the bowels for four months (?).

Sometimes the pressure of these overloaded and meteoristic loops upon the concavity of the liver prevents the escape of the bile, causes jaundice, and makes the fæces light-colored.[1]

Prolonged coprostasis sometimes causes ulcers, especially in the cæcum and flexures of the colon, the so-called stercoral ulcers, which, like those of dysentery, may occasion burrowing suppuration and undermining of the mucous membrane, perforation,[2] or circumscribed peritonitis with stenosis of the canal. The latter also occurs sometimes at the flexures without ulceration, where, when the peristaltic action is weakened, the obstacles to the advance of the contents show themselves first and prominently. Not unfrequently the irritation and stretching caused by the blockaded fæces set up an insidious chronic peritonitis, which may lead to adhesions and narrowing of the intestine. Sometimes the acute circumscribed peritonitis, starting from stercoral ulcers of the colon, plays an important part in the production of impermeability of the intestine; for it is that which, by paralyzing the muscular coat, causes permanent, fatal stasis of the fæces.[3]

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[1] Cases of this kind given by *Bright*, l. c., p. 307; *Nélaton*, in Boys de Loury's excellent work (Gaz. hebd. V. 28, 1858, partly in Schmidt's Jahrb. Bd. 105, S. 328). See also *Wachsmuth*, Virch. Arch. Bd. 23, S. 136; and *Bamberger*, l. c., S. 436, note.

[2] *Dickinson*, Transact. of Lond. Path. Soc. 1868.

[3] *Peacock*, Lond. Path. Soc. 1872.

Intussusception, Invagination, Darmeinschiebung.

Haller, Disp. anat. I. 481; VIII. 97.—*Monro*, Observ. on intussusc. Edinb. Med. and Phys. Essays. Vol. II. 27. III. 388.—*Dance*, Répert. gén. d'anat. et de phys. path. 1829. I. 95.—*Hévin*, Mém. de l'acad. de chir. Vol. IV.—*Langstaff*, Edinb. Med. and Surg. Journ. XI.—*Buet*, Arch. gén. XIV. 230.—*Thomson*, Edinb. Med. Journ. 1835. XLIV. 296.—*Rilliet*, Mém. sur l'invagination chez les enfants. Paris. 1852, and in Gaz. des hôp. 1852.—*Gorham*, Guy's Hosp. Rep. 1838.—*J. L. Smith*, Amer. Journal of Med. Sciences. 1862.—*Rokitansky*, Medic. Jahrb. d. k. k. österr. St. XXIII. N. F. XIV. 554.—*Peacock*, Transact. of the Path. Soc. of Lond. Vol. XV. 1864, p. 113.—*Hansen*, Inaug.-Dissert. Dorpat. 1864.—*Pilz*, Zur Invag. im Kindesalter. Jahrb. f. kinderheilk. 1870. Bd. 3. S. 6.—*Leichtenstern*, Prag. Vierteljahrschrift. 118 and 119. Bd. S. 189; 120. Bd. S. 59; 121. Bd. S. 17.—*J. Hutchinson*, Medico-Chir. Transact. Sec. Ser. London. 1874, p. 31. Furthermore, a mass of theses and journal articles.

Pathological Anatomy.

By intussusceptio intestini we mean the slipping of part of the intestine into the part immediately adjoining. The intussusception is composed of three cylinders; the outer and middle of which oppose their mucous, the middle and inner their peritoneal surfaces to each other. The outer layer forms the intussuscipiens or receiving layer (the sheath, la gaîne, die Scheide), the two middle ones the intussusceptum (le boudin). The innermost cylinder is known as the entering layer (eintretende Rohr); the middle one as the returning layer (austretende Rohr). At the neck (collier, Hals) of the invagination the returning layer turns and becomes the receiving layer (known as the upper or external angle, bourrelet); at the free or lower end of the intussusceptum the entering layer turns and becomes the returning layer (lower or inner angle, le sommet).

From this central or total intussusception we distinguish also a lateral or partial one. It is most frequently formed as follows: a pedunculated polyp, springing from the wall of the intestine, is engaged by the peristaltic action and pressed downwards, and the portion of intestine to which it is attached is drawn after it, like a funnel, into the lumen of the intestine. Velse (De mutuo intest. ingressu. Haller's Disp. anat. T. VII.), as well as van Swieten (T. III. p. 166), was acquainted with this form, which they described under the name of "Volvulus incompletus." [1]

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[1] See my article upon Darm-Invag. l. c. Bd. 118. S. 215.

Between the two cylinders of the intussusceptum lies its mesentery, drawn out and folded in the form of a cone, its apex lying at the lower angle, its base at the neck of the invagination.

The traction exerted by the opposing mesentery upon the intussusceptum produces the following results: 1. That the intussusceptum does not lie centrally in the axis of the enveloping cylinder, but eccentrically, nearer the mesenterial border of the latter. 2. That the intussusceptum is more or less sharply bent with the concavity directed towards the mesenterial attachment, and the convexity towards the opposite side of the receiving layer. 3. That the orifice of the intussusceptum looks towards the mesenterial attachment of the receiving layer, and is distorted into the shape of a fissure. 4. That the cylinder of the intussusceptum, especially in its concavity, presents a more or less deep transverse fold. All these peculiarities are sometimes more, sometimes less marked, and may even be entirely absent, especially in intussusception of the middle part of the ileum.

We distinguish two kinds of invagination, differing greatly in their mode of origin and importance, namely:

1. The "*invagination of the death struggle*," which is an unimportant cadaveric incident, and, as it takes place during the death struggle, or immediately after death, it causes no symptoms during life. It is accompanied by no inflammatory changes, and can be easily reduced; there are often two or more of them at the same time, almost always in the small intestine, never in the cæcum, and are ascending as well as descending. They are seldom found in adults, more often in children, especially such as have died of brain diseases.

2. The so-called "*inflammatory invagination*." This is the only one to occupy us now. Inflammatory invagination takes place, without exception, from above downwards, descending (I. descendens, progressiva), so that a portion of intestine is invaginated into another portion lying below it.

The earlier records contain numerous but in great part very doubtful observations of "inflammatory ascending" invagination (I. ascendens, regressiva). In a collation of five hundred and ninety-nine cases I myself could indicate only eight as such. But even these deserve the name only anatomically, and are essentially different genetically from the primary inflammatory intussusceptions which are always and only descending. These ascending inflammatory invaginations occur at a time when the normal movement of the contents, the normal peristaltic action is arrested by a serious affection of the intestine, usually peritonitis with paralysis of long portions of it. They are, therefore, allied to the invaginations of the death-struggle, and are only distinguished from them by the fact that they appear more promptly, at a time when inflammatory union of the invaginated parts is still possible. On the other hand, in double or multiple invaginations a secondary ascending invagination sometimes occurs together with the primary descending one; this is usually produced by the flaccid, plaited, receiving layer (sheath) of a primary invagination slipping upwards between itself and the intussusceptum. See my work on Darm-Invagination, l. c., Bd. 118. S. 209, et seq.

The primarily invaginated portion, that which enters first, remains at the point of the intussusceptum while the intussusception increases. The growth takes place at the expense of the receiving layer which is turned in to form the middle cylinder. During this lengthening of the invagination the external angle advances downward, and, as is easy to understand, the distance to which the intussusceptum advances within the intestine is double its own length.

The compression and pulling to which the engaged mesentery is subjected causes obstruction, especially of the veins, swelling and sanguineous infiltration of the intussusceptum, and hemorrhages from the mucous surface. Sooner or later peritonitis is set up, and either becomes general and causes death, or remains limited to the intussusceptum, and gives rise to adhesions between the opposing serous surfaces. This makes the invagination irreducible. The portions that are afterwards or consecutively invaginated are often distinguished clearly from the first or primarily invaginated parts by the absence or insignificance of the adhesions and also by the slighter anatomical lesions of the intestinal coats. The condition of the intussusceptum varies according to the course and duration. In acute cases the lesions are limited to the peritonitic glueing together of the invaginated cylinders, and to intense swelling and hemorrhagic infiltration of the coats; in other cases we find more or less extensive grangrene of the intussusceptum, which has broken up into necrotic shreds and fragments. Finally, in chronic cases the closely adherent walls of the intussusceptum are often remarkably thickened, the mesentery transformed into a tough cord of connective tissue that cannot be unfolded, and the muscular coat above the invagination hypertrophied. The serious anatomical lesions are often found exclusively or mainly in the middle cylinder, while the inside and outside ones are relatively or almost entirely unaffected, the latter usually presents no important lesion. Sometimes the mortification of the intussusceptum is especially advanced in the neighborhood of the neck and at the point. Sometimes the separation of the gangrenous intussusceptum takes place very gradually in small shreds, sometimes so insensibly that the process entirely escapes the notice of the patient, sometimes a larger part of the intussusceptum is evacuated, or even the whole invaginated double cylinder (having separated at the neck), and its inner tube may be so well preserved that it is possible to determine the exact seat of the invagination.

The length of the separated piece varies from a few inches to three metres (Cruveilhier).

Intussusception has been observed at all points of the large and small intestine. We distinguish an I. duodenalis, duodeno-jejunalis, jejunalis, jejunoiliaca, iliaca, ileocæcalis, ileocolica, colica, colico-rectalis, and rectalis. Of these only those which occur so often in the region of the cæcum, the ileocæcalis and ileocolica, need a somewhat detailed consideration.

The most common invagination is the *ileocæcalis*—that is, the passage of the ileum and cæcum into the colon, preceded by the ileocæcal opening. The inside cylinder is formed by the ileum. At the apex of the intussusceptum lies the ileocæcal opening, and close by it the opening into the vermiform appendix sometimes very much enlarged.[1] This form, which is also the most common of all chronic ones to disappear, not unfrequently attains a considerable size. Ileocæcal invagination in children, on account of the greater mobility of the colon at that age, often makes its way in a few days through the whole colon, and appears in the rectum immediately above the anus, through which it is even occasionally prolapsed. Another form is that which I have named the *ileocolica.* As in prolapse of the whole rectal wall—for usually the prolapse is composed only of the mucous membrane—the rectum is pressed through the anus and the prolapse is enlarged by the successive addition to the outer cylinder of new portions of the inner one, which were passed through the orifice of the prolapsed double-cylinder,[2] so does it likewise happen in invaginatio ileocolica by the passage of the ileum through the ileocæcal opening. This kind of invagination really represents a prolapse of the ileum through the ileocæcal opening, and the intussusception is enlarged, just as rectal prolapse is—that is, from the apex of the intussusceptum outwards. I annex a diagrammatic sketch of this form, since it has been but little noticed in German literature. (Fig. 17, *C* cæcum, *c* ileocæcal valve, *J* ileum.) It is plain that this kind of increase is possible only so long as the two cylinders of the intussusceptum are not adherent. When this occurs, the enlargement can take place only at the expense of the sheath. First the cæcum and then the colon are turned in, and both make part of the middle cylinder. We have then an ileocolica with secondary cæcum- and colon-invagination, which may also, like an ileocœcalis, pass through the entire colon as far as the rectum. The apex of the intussusceptum is always formed by a longer or shorter piece of the

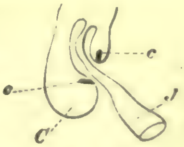

FIG. 17.

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[1] Good plates given by *Weddern*, Dissert. Gryphiæ, 1841. *Blizard*, Med. chir. transact. Vol. I. p. 169.

[2] Cruveilhier calls this form of prolapse of the rectum: Invagination à deux cylindres; Rokitansky: Invagination ohne Scheide (I. without a sheath).

ileum. English literature contains several very close descriptions of ileocolon intussusceptions. A third form of invagination in the region of the cæcum is the *iliaca-ileocolica*—that is, a primary ileum invagination in the lower part of the ileum, which by its increase has passed its intussusceptum through the cæcal opening and into the colon. As this invagination differs from the preceding one by increasing at the expense of the sheath, the outer angle advances towards the cæcum, and finally reaches the ileocæcal opening, as is shown in Fig. 17. It cannot then be determined whether the invagination was primarily ileocolica or iliaca. If the intussusceptum of an iliaca-ileocolica is arrested at the cæcal opening (*e. g.*, by swelling or by spasm of the cæcal sphincter), it may happen, as a result of vigorous peristaltic pressure, that now not only the arrested intussusceptum, but the whole invagination, including the sheath (receiving layer), is further invaginated into the colon. This produces a form of the "*doubled*" or *five-layered invaginations.* Another mode of formation of a doubled invagination is this: that the intussusceptum of an iliaca passes down as far as the ileocæcal opening, and is there arrested by swelling, and now the cæcum, with the ileocæcal opening in front, is pushed into the colon. A third mode of formation is this; the crumpled and relaxed sheath of ileocœcal invagination turns itself, throughout its whole periphery, upwards towards the neck of the primary invagination; and, finally, a fourth kind, when another portion of ileum is invaginated from above downwards into the canal of a primary intussusceptum.[1]

Frequency of Invaginations according to Age, Sex, and Location.

Invagination is by far the most common form of occlusion of the intestines in childhood. Half of all invaginations, according to my statistics of four hundred and seventy-three cases, occur during the first ten years. The first year, after the third month, is remarkable for a special frequency—one-fourth of all intussusceptions. As for sex, it is more common in males than in females at all ages. I. ileocæcalis is by far the most common form of invagination (seventy per cent.) in childhood, especially during the first year. In adults, ileum, and ileocæcal invaginations occur nearly as often.[2] The ileum intussusception is found most frequently in the lower and lowest part of the ileum, most rarely in the middle portion. Colon invaginations are more frequent in the descending colon and sigmoid flexure than in any other portion.

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[1] For further details of manifold invaginations see my work, l. c., S. 210, et seq. Bd. 118.

[2] My statistics of four hundred and seventy-nine invaginations, at different ages, gave fifty-two per cent. of ileocæcal and ileocolon, thirty per cent. of ileum, and eighteen per cent. of colon invaginations. The relative frequency of ileocæcal and ileocolon invaginations cannot yet be determined by statistics, for attention has but seldom been given to distinguishing them from one another.

Etiology and Pathogenesis.

Since the time of J. C. Peyer,[1] who first separated intestinal invagination from the general expression, volvulus, embracing different things, its mode of origin has been explained as due to the forcing of a portion of intestine possessing normal or increased peristaltic action into a portion lying below it, and paralyzed by violent diarrhœa, ingesta, traumatism, or partial peritonitis ("I. paralytica"). The process has always been represented to be such that the paralyzed portion becomes the sheath of the invaginated cylinders. But it seems to me there are weighty reasons for thinking that at an early period, often at the very beginning of the invagination, the whole paralyzed portion is turned in and is invaginated with the penetrating innermost cylinder into the normal intestine lying below the paralyzed portion, and it is this normal part which forms the sheath. In this way is explained the fixation of the intussusception when it has once penetrated, a fixation which, when we consider the opposition exerted by the invaginated mesentery, presupposes an active participation by the sheath, a sort of tenesmus of it. It also explains the compression of the intussusceptum and its mesentery, which sometimes leads to swelling, bloody infiltration, and peritonitic adhesions of the invaginated cylinders; it explains the circular constriction of the intussusceptum at the neck. The clinical course also corresponds best with this explanation. The sudden appearance of invagination with violent colics corresponds to the moment when the paralyzed portion, turned in and driven by the inside cylinder, which is still capable of peristaltic action, becomes invaginated in the active intestine below; the paroxysmal colics are caused by the paroxysmal tenesmus of the sheath. The rapid growth, too, of many acute invaginations can only be thus explained, that the peristaltically potent sheath forces the intussusceptum, like anything else contained in it, in the normal direction downwards. The increased peristaltic action expresses itself also symptomatically in the diarrhœa, which is so common in intussusception. After what has been said, no further explanation is needed to show that paralysis, when limited to a small piece of intestine, is more favorable to the occurrence and fixation of an invagination than when the affected portion is longer. *Paresis of a limited portion of the intestine, associated with vigorous peristaltic action, excited by any cause whatsoever, offers suitable conditions for invagination.*

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[1] Parerg. anat. et med. septem. 1677. Lugd. Bat. 1736, p. 61, et seq.

With reference to the origin of invagination in paralysis of a portion of the intestines, I cannot refrain from calling attention to the following circumstances. We have already seen that in dysentery, according to Annesley, Griesinger, and others, paralysis of a section of intestine, with consecutive severe fæcal stasis (without stenosis), is not uncommon. It is interesting that intussusceptions have also been repeatedly observed in the course of chronic diarrhœas. Griesinger, for example, reports three cases of this kind (Ges. Abhdlg. II. 686). I also have observed that a traumatism of the abdomen may cause death by ileus, and that often we cannot explain these cases otherwise than by the supposition of paralysis of a large portion of the intestine, causing complete interruption of the normal movement of the contents. A number of most trustworthy observers have reported cases of invagination which occurred in previously healthy individuals immediately after a traumatism. Also in those invaginations which occur, though very seldom, after typhoid fever, cholera, with tubercular intestinal ulcers (without stenosis), with tubercular peritonitis, or after the reduction of a strangulated hernia, the paralysis of the corresponding portion of the intestine should be considered of etiological significance. Finally, I must mention that invagination of the rectum (with or without prolapse) arises under the same conditions of relaxation of the rectum and its attachments (after chronic blennorrhœa or prolonged tenesmus) as does the ordinary prolapse of the rectum composed only of mucous membrane.

Another early theory of the formation of intestinal invagination was, that it was due to the tetanic contraction of the circular muscular fibres of a short portion of the intestine, in consequence of which the latter is forced by the peristaltic action of the portion lying above it into that which lies below, and is then firmly grasped ("invag. spasmodica"). Presented in this way, the theory is scarcely correct; but, as the following will show, there is a certain foundation for it.

The fact that invagination occurs so much the more frequently in the region of the cæcum and in the lowest portion of the ileum immediately above the ileocæcal valve, has always invited attempts at explanation. Special importance has been attached to the greater breadth of the cæcum and colon into which the smaller ileum could easily enter, also to the more fixed position of the colon, which forms the sheath, as compared with the greater mobility of the penetrating ileum rendered possible by its mesentery. The greater sluggishness, too, of the former has been invoked, and the more vigorous peristaltic action of the latter.

The importance of these factors is certainly not to be underestimated, but the principal cause is to be found elsewhere. If we consider that the ileocæcal opening is distinguished by a sphincter, the contraction of which can increase to powerful tenesmus, we recognize that there is a complete analogy between the conditions of invagination in the region of the cæcum and the different kinds of prolapse of the rectum, which, like ileocæcal invaginations, is found most frequently in early childhood. Just as anal tenesmus, excited by any cause whatever (rectal blennorrhœa, profuse diarrhœa), usually excites and accompanies prolapse of the rectum, so is ileocæcal tenesmus, excited by catarrh or abnormal irritability of the terminal portion of the ileum, of great importance in the production of many ileocæcal and ileocolon invaginations. In many cases in which we see invaginations in the region of the cæcum follow prolonged diarrhœa or colic, the taking of unsuitable food, or, especially in early infancy, the withdrawal of the mother's milk and the substitution of improper food, cæcal tenesmus plays an important part. If the cæcum and the colon are rendered easily movable by their mesentery—as is regularly the case during early life—the repeated and more forcible peristaltic pressure towards the persistently contracted ileocæcal sphincter causes ileocæcal invagination. If the cæcum is firmly fastened down, so that it cannot be turned in and invaginated into the colon, prolapse of the ileum into the colon takes place, with formation of an ileocolica invagination, just as prolapse of the rectum may follow violent anal tenesmus. If neither of these happens, invagination of the lowest part of the ileum may occur, as is the case also in the rectum when it becomes invaginated in itself above an obstinately contracted (tenesmus) sphincter, and is finally prolapsed (called by the surgeons "prolapse of the rectal wall with invagination").

If ileocæcal invaginations are very common in children, and ileum invaginations, on the contrary, very rare, the reason lies in the greater mobility of the cæcum and ascending colon allowed by their mesentery, and the consequent removal of an obstacle to invagination. In adults this element is removed, and we find ileum invaginations as frequent as ileocæcal. Probably a not uncommon abnormal mobility of the cæcum and ascending colon is an element in the production of the latter in adults also.

An important part in the etiology of invaginations is taken by neoplasms of the intestinal canal, especially such as have a pedunculated attachment to the mucous membrane and project in the form of a polyp into the lumen of the canal. The records are full of cases of invagination due to polyps. Since the polyp is grasped by the peristaltic action and forced towards the anum like the rest of the contents, it draws the intestine from which it springs as an intussusceptum behind it.

On the other hand, it is exceedingly uncommon for circular stenosis of the canal to cause invagination, for the reason that the introduction of the voluminous intestine above the stenosis, preceded by the stenosed part, into the usually contracted portion lying below it, meets with mechanical obstacles. Where these do not exist, as in stenosis of the ileocæcal opening, we find that intussusception is not uncommon.

Among the incidental, and in part also indirect causes, a well-ascertained part, according to the most trustworthy observations, is played by improper ingesta, traumatism of the abdomen, violent jarring of the body, prolonged diarrhœa, colics, and constipation. The mode of action of these causes can be easily made out by the aid of the foregoing.

It must be remarked that the symptoms of invagination of the intestines, especially in children, appear suddenly in the majority of cases, without being preceded by any known cause or disturbance of health, a fact which deserves all the more notice because an improper importance has been attributed by many to prodromal chronic diarrhœas, colics, etc., as the regular forerunners of invagination.[1]

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[1] For details, see my statistical communications, Prager Vierteljahrschrift, Bd. 118 and 119, S. 66, S. 72, et seq.

In earlier times, when intestinal worms were supposed to be the cause of many diseases and morbid symptoms, they were often held responsible for invagination of the intestine also. There is no support for such an opinion. Sometimes the ascarides, as Ruysch described and De Haën[1] pictured it, wind themselves about the intussusceptum, an entirely unimportant incident which can have no connection with the etiology for the reason that an intussusception must be present before the ascarides can wind themselves about it.

Symptoms, Course, Duration.

The symptoms of invagination of the intestine usually appear suddenly, whether an incidental cause has preceded it or not. The scene opens with a *violent colic.* Then follows *vomiting*, and this occurs promptly, especially in children and when the invagination is situated high up.

After a longer or shorter time, often after a few hours in children, the pains cease, and now several hours may pass during which the serious affection betrays itself by no sign, until a new paroxysm of pain occurs. In adults the pain introducing the invagination often lasts continuously for several days, and then the colics alternate with intervals of freedom from pain. The intensity of the pain is often such as is found in only a few affections of the intestines. In childhood invagination is sometimes introduced by general convulsions.

The peristaltic action, at times very violent, which is excited by the invagination, causes a rapid evacuation of the contents of the intestines, a *diarrhœa* which amounts often to from ten to twenty passages in the twenty-four hours. The compression of the veins of the invaginated mesentery causes venous stasis and swelling of the intussusceptum, hemorrhages by diapedesis and by rupture, and often abnormal secretion of mucus as a result of catarrh of the mucous membrane. The so-called *dysenteric evacuations*, consisting of *bloody mucus*, are almost never absent in invagination of the intestines, no matter where its seat may be.

This symptom is the more important, because, with the exception of twisting and knotting of the sigmoid flexure, no other cause of acute occlusion of the intestine is accompanied by bloody evacuations. On the other hand, attention must be paid to the differential diagnosis from dysentery and from the other sources of intestinal bleeding—especially rectal polyps in children.

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[1] Rat. med. contin. T. II. P. II. Tab. II.

The profuse diarrhœa leads at once to violent *tenesmus ad anum*, which appears the earlier and is the more intense the nearer the intussusceptum was originally to the rectum, or is brought to it by its growth. When the intussusceptum occupies the descending colon or the rectum, the tenesmus is not unfrequently followed by *paralysis of the rectal sphincter* and patulence of the anus, through which the bloody diarrhœal passages escape involuntarily, a symptom which is found especially in children.

A patulous anus is certainly not pathognomonic of invagination of the intestine, as some have believed. The same thing happens in diarrhœa sometimes, often with neoplasms in the sigmoid flexure or rectum,[1] even with foreign bodies, and in individuals suffering from paraplegia, etc.

The *meteorism* which appears immediately after the occurrence of invagination disappears with the subsidence of the acute swelling of the intussusceptum and with the following diarrhœa. In the subsequent course of subacute and chronic invaginations meteorism is a very variable symptom. A far more important symptom is the usually *cylindrical, sausage-like tumor* which can be felt *in the abdomen* in most cases of invagination. *This can almost always be felt in colon and ileocæcal invaginations, but only seldom in ileum invaginations.*

Since ileocæcal invagination is the usual one in children, it is evident that at this age the tumor is but seldom missed. The invagination tumor is found most frequently in chronic cases, more rarely in acute ones. In ileocæcal intussusception it is found only exceptionally in the region of the cæcum, but often in the transverse or descending colon and in the regio iliaca sinistra. In chronic ileocæcal invagination the position of the tumor often changes, and it is found sometimes in the transverse colon, sometimes further along in the descending colon; the invagination increases in such cases by the turning in of new portions of the sheath; by the reduction of these freshly invaginated, non-adherent portions the tumor returns to the transverse colon. This change in the length and position of the tumor may be observed repeatedly within a short time. In ileum invaginations the hypogastrium is sometimes the seat of the tumor. An invagination tumor remaining stationary for a long time in the region of the cæcum indicates ileocolica. The tumor shows still other peculiarities. It often seems to be absent at the beginning of the search. After some palpation and handling it becomes more distinct, firmer, and easier to grasp in all directions. Moreover, it often happens that while the tumor cannot be found during the intervals that are free from pain, it presents itself as a stiff cylindrical roll during the paroxysms. Sometimes in ileocæcal and colon invaginations the tumor can ultimately be felt in the rectum, or it may even be prolapsed through the anus, and the certain diagnosis of the ileocæcalis can be made during life by the sight of the two orifices—the ileocæcal and that of the vermiform appendix—at the apex of the intussusceptum. The increase of ileocæcal intussusception is often very rapid in children, so that the intussusceptum may reach the rectum by the second day of the attack. The cause of this lies in the great mobility of the child's colon distinguished by a long mesocola.

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[1] *Sölversberg*, Schmidt's Jahrb. 80, Bd. S. 208. *Rokitansky*, Oesterr. med. Jahrb. X. 1836.

We have now to speak of the course and terminations. We distinguish:

The *acute course.* The invagination leads to immediate and permanent impermeability. The patients die in from three to six days with the symptoms of internal strangulation (with or without peritonitis or perforation). This course belongs chiefly to ileum invaginations, especially such as are situated high up in the small intestine. Another kind of hyperacute course is death by shock, especially in children less than a year old.

In other cases the permeability of the invagination is restored by the reduction of the swelling of the intussusceptum, profuse diarrhœa sets in, but death follows from prompt gangrene of the intussusceptum which gives rise to general peritonitis or perforation. In children the fatal termination in this way generally takes place between the fourth and seventh days, in adults in the course of the second week, but sometimes, when subacute, not until the third or fourth week. Since the gangrenous process is not always equally advanced at all points, *the separation of the intussusceptum*[1] often takes place fragmentarily in single strips, shreds, or tubular sections, and sometimes so gradually that it is entirely overlooked by the patient. In other cases the intussusceptum separates over the entire periphery of the neck of the invagination, and is evacuated entire, in which case the inside cylinder, and sometimes both, presents a still well-preserved appearance, while the line of separation is gangrenous and shreddy. *The separation occurs, in the majority of cases, from the eleventh to the twenty-first day* after the production of the invagination, but in chronic cases it may not happen until after several months. Separation takes place most frequently in ileum invaginations. It is exceedingly rare in children under two years of age, for they usually die before it can take place. Numerous trustworthy observations show that separation of the intussusceptum may lead to complete cure of the invagination.

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[1] Many of the earlier accounts of "intestinal invagination," with separation of "gangrenous portions of the mucous membrane" or "tubular pieces of intestine," depend upon misapprehension, for the supposed pieces of intestine were nothing but undigestible ingesta (skin, intestines) or intentional admixtures. (*Virchow*, in Virch. Arch. V. 302). In the severe diarrhœas of the tropics portions of the mucous membrane of the colon often come away, as the best observers (Annesley, Griesinger, and others) have testified.

But this mode of termination has many dangers which threaten life. The separation may take place too early, before there is solid union at the neck of the invagination. Perforation, or rather rupture, is the result; or a part of the intussusceptum remains, and causes a new invagination by increase of what is left of the old one. In other cases constriction of the intestine takes place at the circular line of separation, increases more and more, and often causes death after some years. Finally, torpid ulcers of the mucous membrane may remain at the place of separation and cause perforation long afterwards, or defective nutrition, as a result of constant diarrhœa; in which latter case the patients die in marasmus, with dropsical symptoms, after long confinement to bed. Among the rarer terminations, after separation of the intestine, we find death by septicæmia, due to the absorption of putrid elements at the ulcerated point of separation; and thromboses, which extend from the point of separation to the large mesenteric veins, and give rise by embolism to metastatic abscesses in the liver, and in the lungs also under certain circumstances. Perforation into the retro-peritoneal cellular tissue causes its destruction and the formation of a fæcal cloaca. In this way inflammation and suppuration of the psoas may be set up, just as in perityphlitis, and, as a result, the movements of one of the lower limbs may be limited. As the inflammation extends to the nerves of the plexus cruralis, lying partly within the psoas and partly between it and the iliacus, it causes neuralgia and anæsthesia, paralysis, and contractures in the regions supplied by branches from this plexus.

The progressing separation of the intussusceptum is often indicated by the extraordinary foulness of the evacuations, and by their admixture with blood and gangrenous shreds of intestine. Not unfrequently the separation is immediately preceded by an obstruction lasting for several days, with notable aggravation of the general condition (meteorism, collapse, vomiting—even stercoraceous vomiting). The intestine is then suddenly opened; profuse fetid evacuations indicate the loosening at the neck of the invagination and the freeing of the passage, and after one or more days the separated intussusceptum is evacuated per anum. The passage of this intestinal sequestrum is sometimes a hard task. It has in some instances caused total obstruction at a lower point, and many ruptures at the line of separation would not have taken place if the intestinal sequestrum had not aided by occluding the passage below.

We also distinguish the *chronic course.* This is by far the most frequent in ileocæcal invaginations, and is due to the fact that the invagination becomes fixed by the solid union of the two cylinders, and the canal of the intussusceptum becomes again permeable by the complete disappearance of the swelling. In this way the ileocæcal invagination may last for several months. In one of the cases observed in Liebermeister's Clinic and described by me,[1] the accurately determined duration of the invagination was eleven months; and we have even trustworthy records of cases—cases which in my opinion are not difficult to understand—in which the intussusception lasted four, three, two, or more than one year, and ended in recovery by separation, or in death by perforative peritonitis, or by increasing marasmus, with chronic diarrhœa. If the invagination becomes chronic, the diarrhœa usually moderates, the evacuations no longer contain blood, and they may even become completely normal again. In other cases severe constipation alternates with profuse diarrhœa, or the latter becomes chronic and seriously affects the nutrition of the patient. The most important symptoms—those which indicate the continuation of the intussusception—are the paroxysms of colic, usually occurring daily, sometimes more than once each day, and the presence at some point in the colon of a tumor, usually cylindrical, which not unfrequently presents the peculiarities before described. Since in these cases the muscular coat above the invagination becomes hypertrophied, it often happens that the outlines of the convolutions are visible through the emaciated abdominal walls, and indicate the existence of a chronic obstruction.

Finally, we have yet to mention among the terminations of invagination *recovery without separation.* It has been firmly established by numerous communications from famous authors that intestinal invaginations may be reduced spontaneously or by suitable treatment (opium, enemata, injections of air, etc.). The correctness of the diagnosis cannot be doubted when, as is not unfrequently the case, the intussusceptum has been touched in the rectum or seen, after being prolapsed, through the anus. Cruveilhier and Gorham have expressed the plausible opinion that invaginations often occur and are again reduced, and that, in particular, many of the temporary severe colics in children depend upon this cause.

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[1] Deutsch. Arch. f. klin. Med. XII. Bd. S. 381, et seq.

Differential Diagnosis of the Different Kinds of Occlusion of the Intestines.

Now that we have considered the symptoms, course, and terminations of the different kinds of occlusion of the intestines, we may turn our attention to their differential diagnosis.

In most cases of internal occlusion of the intestine, our diagnosis is limited to determining its existence, and perhaps, also, its probable seat, and we are not able to do more than form a supposition regarding its anatomical cause. Even those processes of occlusion which are distinguished under favorable circumstances by definite peculiarities and objective signs, and make an absolute diagnosis possible, sometimes occur without them, and are then not to be distinguished from other causes of impermeability. Thus, to give a single example, the records contain many cases of neoplasms of the intestines which have remained completely latent and then have suddenly caused death with symptoms of acute internal strangulation. In many cases decisive information is furnished by close attention to the beginning and progress, the duration and symptoms, the seat of the occlusion, the age and sex of the patient, also by a thorough examination, and, finally, by the antecedent history. Under such circumstances an absolute diagnosis is sometimes possible, and sometimes we are at least able, by exclusion, to limit the possibilities to a few, and by attention to the relative frequency of these to make a diagnosis more or less well founded upon probabilities. Let us briefly consider those occlusions which, under favorable circumstances, we are able to diagnosticate certainly or with probability, by the aid of objective or anamnestic information.

In the first rank stand those occlusions which are situated in the rectum and accessible to direct examination by palpation, sometimes, also, by inspection. Here belong serious fæcal obstructions of the rectal ampulla, such as are seen especially in advanced life; compression of the rectum by tumors arising from the organs and walls of the pelvis, occlusions which succeed to a constriction that has lasted a long time, and the nature and cause of which were known, and, finally, occlusion by foreign bodies and intussusceptions which have made their way to the rectum.

Anamnesis often furnishes an absolute diagnosis in (total or partial) obstruction by foreign bodies that have been swallowed. Enteroliths are usually situated in the rectum, where they repeatedly give rise to symptoms of typhlitis and diminished permeability, and furnish evidence of the constant presence of a tumor in the region of the cæcum before occlusion comes on. The diagnosis can be made with certainty only when pieces of the stone have been evacuated (Monro's case).

I agree with Habershon, Brinton, and others, in thinking that the diagnosis of obstruction by gall-stones is possible under favorable circumstances. The appearance of symptoms of occlusion immediately after a severe attack of hepatic colic accompanied by icterus, sometimes with an accurate history of previous attacks of hepatic colic, perhaps the passage of gall-stones, or the rare occurrence that a small gall stone is vomited during the ileus (Merly), attention to the age and sex (vide supra), the seat of the occlusion (uppermost part of the ileum, duodenum, jejunum, or neighborhood of the ileocæcal opening), all aid in the diagnosis.

Occlusion of the intestine by fæcal masses (I. paralyticus), is usually preceded for a long time by habitual constipation, with occasional evacuations of what are often enormous masses of fæces. It occurs most frequently in elderly people. The repeated finding of fæcal tumors in the abdomen, perhaps, too, a previous attack of ileus, which got well after the evacuation of a large amount of fæces, the examination of the rectal ampulla, the exploration of the colon by injections, the location in this way of the seat of the occlusion in the colon, often establish the diagnosis, although it is usually impossible to say with certainty whether the impermeability depends solely upon functional causes, or whether there is, in addition, a constriction at some point in the colon. We have already mentioned that not unfrequently another factor is added to fæcal obstruction (peritonitis, twisting of the loop filled by the fæces, kinking by falling into the pelvis, compression by the mesentery of an overlying portion of the ileum), by which the obstruction is produced and the symptoms of acute internal strangulation excited.

As for occlusion of the intestine by compression, the physician and patient are not unfrequently aware, before the occurrence of the occlusion, of the presence of a tumor in the pelvis or abdominal cavity, or it is found during the exploration of the abdomen, vagina, or rectum. Occlusion by twisting of the sigmoid flexure, by its compression by a loop of the ileum, and the formation of a knot between it and the ileum, are all recognized by their acute beginning and rapid course with all the symptoms of internal strangulation and by early collapse. Enemata and insufflation of air (especially with simultaneous auscultation) show the lower part of the colon to be the seat of the occlusion. The formation of a knot between the sigmoid flexure and ileum is very often accompanied by bloody diarrhœa. Habitual coprostasis usually precedes, for a long time, occlusions of this kind, which are found most frequently in men over forty years old; diarrhœa immediately introduces them.

Among occlusions which are very often recognized during life belong intussusceptions. The sudden beginning of the attack with violent intermitting colics, the bloody mucous diarrhœa with tenesmus, the appearance, increase, and change of place of a sausage-shaped tumor presenting the characteristics above-mentioned, the possibility sometimes of touching the intussusceptum in the rectum, or of seeing it prolapsed through the anus, the evacuation of gangrenous shreds of the intestine, or of a well-preserved cylinder, and attention to the age—intussusceptions are relatively frequent in childhood, and all other causes of occlusion are very rare at that age—often allow a more or less absolute diagnosis. In many cases this would be established if a simple examination of the rectum were made. With reference to the differential diagnosis in children, especial attention must be given to simple severe colics, rectal polyps with bloody diarrhœa, and dysentery. As for the latter, the early appearance of the tumor in invagination, the sudden attack, and the course, are especially of diagnostic significance, Intussusception in the highest part of the ileum or jejunum, usually mocks at diagnosis, since it has none of the characteristic signs of ileocæcal or colon invagination.

If the occlusion is superadded to the symptoms of a long-standing constriction of known character, its ultimate cause is thereby made known. On the other hand, its immediate, direct cause remains unknown—whether paralysis of the intestine above the stenosis, with arrest of the fæcal current, or a superadded kinking, or twisting or obstruction of the stenosis by a hard piece of fæces or foreign body.

We have finally to mention a number of causes of occlusion which do not allow a definite diagnosis, because they are exactly alike in their commencement, course, and symptoms, and because the anamnesis also can supply no information. These are strangulation by false ligaments, omental bands, the vermiform appendix, a diverticle, strangulation in fissures and holes in the omentum, mesentery, or other viscera. We can also mention twisting of an adherent loop of the ileum, compression of the lowest part of the ileum or of the bent cæcum and ascending colon, displaced towards the median line, by the mesentery of a loop of the small intestine. All these conditions have, in common, besides the sudden beginning and rapid course, the fact that examination of the colon by enemata shows its permeability, and also that all the other symptoms indicate occlusion of the ileum or cæcum.

But of the varieties of occlusion previously considered, which, under favorable circumstances, allow a diagnosis based upon certain objective and commemorative points, many behave exactly like those last mentioned, and can in no way be distinguished from them. Our diagnostic ability in such cases is limited to being in a position to say that one kind of occlusion is improbable, and that another, relying upon their relative frequency, is more probable. Thus, for example, when the symptoms of internal strangulation appear in a woman, we are less inclined to think of strangulation by a diverticle; if the patient is more than forty years old, strangulation by a diverticle is, according to our statistics, very improbable. On the other hand, in a patient under twenty years of age, we should consider strangulation by a false ligament, or the appendix vermiformis, as improbable; at this age strangulation by a diverticle is most common. In patients who have previously suffered from peritonitis, or typhlitis, especially in women who have borne several children, or have had pelvic peritonitis, strangulation by a false ligament has a certain degree of probability. Before even a probable diagnosis of one of the kinds of incarceration under consideration is made in a child under ten years of age, examination should have established the absence of an intussusception.

Of the internal hernias only the diaphragmatic can sometimes be recognized by the signs previously mentioned.

By careless attempts at taxis an external hernia may be transformed into a retroperitoneal internal one; the sac is loosened at all points and forced, with its contents, into the subperitoneal layer between the peritoneum and transverse fascia. If the symptoms of strangulation persist after such taxis the diagnosis of reduction in mass is sometimes rendered possible by a series of symptoms which are fully described in surgical books. In other cases, again, it remains uncertain whether there has been reduction in mass, or passage of the contents of the sac into a previously existing interstitial or subperitoneal pocket, or through a tear in the sac into the inter-muscular or subperitoneal cellular tissue, or if the duration of the symptoms, after successful reduction, is not due to kinking, torsion, or paralysis of the intestine that had been strangulated.[1] If we learn from a patient with internal strangulation that he has previously had a hernia reduced, which has not since reappeared, or if he has at the time a reducible external hernia, there is a very strong probability that the latter, or its surroundings, have some connection with the internal strangulation. There may be strangulation of an anterior retroperitoneal hernia, due to reduction in mass, or in an interstitial hernial pocket, or by omental bands, since they are very likely to become adherent in hernia.[2] But there are still other possibilities. A loop of small intestine, attached near the hernia, may become twisted[3] about its mesenterial axis, or the loop which was formerly contained in the hernia may have become considerably stenosed by a progressive chronic peritonitis, and this stenosis may have caused the occlusion. I must here speak of a circumstance which deserves attention. If the intestine, for any cause, becomes impermeable below an external hernia, the latter shares in the distention of the portion lying above the obstruction, it is driven more forcibly into the hernial sac, can be reduced with difficulty into the tense, meteoristic abdomen, and thus may look as if it were itself strangulated, and the cause of the impermeability. Lucke and Ward[1] cut down upon the hernia in such a case, and then enlarged the incision and performed laparotomy. The autopsy showed a stenosis of the colon twelve inches above the anus. Borelli[2] relates a similar case.

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[1] *Streubel*, Schmidt's Jahrb. Bd. 110, S. 365.

[2] *Monro*, Morb. anat. p. 536. *Renould*, Journ. gén. de méd. T. XVII. p. 24. *Dupuytren*, l. c. *Fagge*, l. c., p. 352. *Streubel*, l. c., S. p. 363.

[3] *Ruehle* and *Busch*, Sitz. der med. Sect. d. niederrhein. Gesellsch. in Bonn. 15 Mch. 1865. *Fagge*, l. c., p. 352. *Froriep*, Chir. Kupfertafeln. 370. VIII. 178.

Constrictions of the Intestines.

Cicatricial Stenoses.

The different processes which give rise, in this way, to constrictions of the intestine, are:

Dysentery. The deep lesions of the mucous membrane and submucosa in the diphtheritic form of epidemic or sporadic diarrhœa lead, during the often prolonged process of recovery (chronic diarrhœa), to marked stenosis, not unfrequently at several points.

Unaffected islands of mucous membrane often persist between the diphtheritic losses of substance, which are made to project into the lumen of the canal in the form of knobs and folds, by the contraction of the adjoining cicatrices (*valvular stenosis*). By the contraction of unilateral cicatrices the intestine becomes bent; when the cicatrix is extensive and on all sides, the intestine is drawn together in the direction of its longitudinal axis, and thus stiff, callous folds, bands, and sickle-shaped projections into the canal, lying one above the other, are produced (picture in Albers, Tab. XIII.). Constrictions of this kind are often increased by tough polypoid excrescences[3] growing from the edges of the mucous membrane into the canal, and which sometimes act like valves and increase the stenosis.[4] If the process becomes localized, as happens exceptionally (Annesley), especially in the cæcum, at the cæcal valve, and in the lower part of the ileum, this portion of the intestine sometimes becomes transformed into a stiff mass of intestinal cicatrix drawn up into folds, which scarcely allows its anatomical nature to be recognized. Corresponding to the usual localization of the diarrhœal process, the descending colon, sigmoid flexure, and rectum are the most frequent seat of the constriction.

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[1] Lond. Path. Soc. Vol. II. 218.

[2] Gaz. med. d'Ital. 1854.

[3] Often described formerly as "scirrhus." *Meckel*, Entr. Comment. Leipz. T. XV. *Monro*, l. c., p. 155. See *Habershon*, l. c., p. 385.

[4] Cases have even been reported of complete union (?). *Monro*, Morb. Anat. p. 155. *Rhodius*, Mis. Cur. Obs. 508.

It is especially at the flexures of the colon that the stenoses resulting from dysentery are most frequent and most severe. Dysenteric stricture, for the reasons already given, is one of the most severe. And yet tight strictures, found at the autopsy in not a few cases of chronic diarrhœa in which death has occurred after many years of illness, have caused no noticeable symptoms during life, and for this reason, that the continuation of the destructive process, aided, too, by the stenosis, has kept up a chronic catarrh, which has prevented the contents of the colon from acquiring the consistency of normal fæces, and has excited diarrhœa, and diarrhœal discharges can make their way through notably constricted passages. It may be laid down as a rule that symptoms are manifested earlier by dysenteric stenoses the sooner the colon returns to its normal condition. Still, the symptoms of constriction may first appear only many years—thirty years in a case of Rokitansky's—after the attack of dysentery, and then cause death by occlusion.

Stenoses which are *caused by the cicatrization of tubercular ulcers* of the intestine very seldom attain a notable degree as the result of a simultaneous constricting peritonitis at the ulcerated point. They are commonly situated in the lower part of the ileum ; sometimes, though rarely, in the rectum. One should not infer the existence of stenosis in a case of tuberculosis of the intestine because the convolutions and their motions can be seen through the thin abdominal walls.

I refer to the surgical text-books for accounts of constricting ulcers in the rectum which proceed from catarrhal proctitis, with or without formation of a fistula, from follicular or hemorrhoidal ulceration, from virulent blennorrhœa, or are caused by wounds, foreign bodies, etc. Syphilitic ulcers (hard and soft chancres, condylomatous ulcerations, or tertiary ulcers produced by the breaking down of gummy infiltrations) occur but seldom in the rectum, and lead still more rarely to stenosis. Only very few cases are known of gummy infiltration of the mucosa and submucosa of the small intestine which have given rise to ulceration and, by cicatrization, to constriction of the intestine.[1] Among the ulcers which in rare cases cause stenosis by their cicatrization, but in which the accompanying constricting peritonitis plays an important part, I mention stercoral ulcers in the colon (especially at the flexures), which are caused by the mechanical and chemical irritation of arrested fæcal masses ; also constrictions of the cæcum or ileocæcal opening, following chronic stercoral typhlitis ; and, finally, ulcers caused by foreign bodies, separated intussuscepta, or enteroliths. Extensive, spreading, confluent typhoid ulcers, also, in the cæcum, ileocæcal valve, and lowest part of the ileum, have also in rare, exceptional cases been seen to be the starting-point of stenosis (Klob [1]); also a round ulcer of the duodenum in very rare cases. Finally, it may be mentioned that cicatricial stenoses have been repeatedly found in the large and small intestines, of which no satisfactory explanation could be furnished by the history of the case or the appearance of the scar.

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[1] See *Klebs*, Path. Anat. 2 Lief. S. 261, et seq. ; *Esmarch*, in Pitha-Billroth's Handb. S. 85. Further : *Sauri*, Thèse de Paris, 1868 ; *von Bärensprung*, Charité-Annal. Jahrg. VI. ; *Probst*, Diss. Inaug. Berlin, 1868 ; *Oser*, in the Unters. d. path.-anat. Inst. in Krakau, 1872.

Stenoses Caused by Chronic Peritonitis.

(Constrictions, Adhesions, Chronic Inflections.)

We have repeatedly spoken of the important part which partial, chronic peritonitis plays in the different processes of internal strangulation. We saw that in many cases the intestine has been long adherent to the cause of the strangulation, *e. g.*, to a diverticle, false ligament, omental band, or to the hernial orifice in internal hernia, and not unfrequently is itself constricted by chronic peritonitis, until at last the condition leads, usually suddenly, to strangulation. On the other hand, it sometimes happens that an incarceration or compression that has begun acutely produces only temporary total occlusion, and that a period of constriction follows that of occlusion, produced by the adherence of the intestine to the constricting or compressing cause. In both cases we speak of chronic strangulation, chronic torsion, chronic compression, etc. We saw that many cicatricial strictures of the intestine were accompanied by a constricting peritonitis, which had an important part in the production of the constriction. The adhesions produced by chronic, circumscribed peritonitis between the intestine and another part of itself, or tumors of different kinds, or the organs and walls of the abdominal and especially of the pelvic cavity, the shrinking of tubercular mesenteric glands and the corresponding mesentery, the chronic peritonitis after typhlitis and perityphlitis, the adhesion of the omentum to the intestine, and many other causes lead partly by constriction and partly by kinking and traction to manifold narrowing of the lumen of the canal. We should be forced into many repetitions if we tried to describe separately the processes of chronic peritonitis which participate so often and in such various ways in the production of constrictions of the intestine. We shall confine ourselves to the description of the following, more independent processes.

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[1] Wien. med. Wochenschr. 1863.

1. *Chronic general peritonitis* (simple, tubercular, sarcomatous, carcinomatous) not unfrequently gives rise to manifold adhesions between the intestinal loops, to numerous bends, constrictions, torsions, and tractions of the intestine. We often find the entire small intestine matted together into a sort of homogeneous mass, so that it is only with difficulty that the scissors can make their way through the labyrinth of bends and curves. In such cases we may have all the symptoms of diminished permeability and sometimes of absolute occlusion.[1] A few weeks ago I saw a case of this kind in which obstruction, lasting ten days and followed by another of fifteen days, preceded death, which took place after constant vomiting, anuria lasting for several days, aphonia, cramps in the legs, subnormal temperature, with extreme dessication of the patient.

2. An intestinal loop which has lain for a long time in a hernia sometimes becomes the seat of an insidious, constricting peritonitis, which leads gradually to increasing entero-stenosis, as a consequence of many injuries by ill-fitting trusses, frequent attempts at taxis,[2] etc. Not unfrequently this process first appears within the abdominal cavity after the successful reduction of a strangulated loop. Adhesion, bending, constriction, and conglomeration,[3] or chronic torsion of the replaced loop may result therefrom.

3. A circumscribed, chronic, constricting peritonitis is sometimes found at the flexures of the colon. As the results of atony of the muscular coat repeated fæcal accumulations are

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[1] *Corbin*, Arch. gén. de méd. T. XXV., I Ser. 36, XXIV. I. p. 214. See page 586.

[2] *Blöst*, Schmidt's Jahrb. V. 82. Several interesting cases are recorded by *Palasciano* (l. c.), *Reuter*, *Gruber*, and others. See: *Streubel*, Schmidt's Jahrb. Bd. 110, S. 364, et seq. *Birkett*, London Path. Soc. X. 128. *Bristowe*, ibid. X. *Stein*, Memorabil. Heft 6, S. 141. *Fagge*, l. c. S. 323 and 352. *Froriep*, Chir. Kupfertafeln. 370, VIII. 178. See especially *Moraweck*, Schmidt's Jahrb. Bd. 85, S. 330, and *Ernest Guignard's* valuable work, Thèse Inaug. Paris, 1846.

[3] *Rokitansky*, Oesterr. med. Jahrb. X. 1836. *Godin*, Bull. de la soc. anat. 1836, p. 301.

found especially at the flexures, the points where the obstacles to the advance of the fæces are greater. The frequently repeated irritation of the peritoneum produced thereby excites chronic peritonitis, which may result in constriction. In other cases the chronic peritonitis starts from the concavity of the liver and extends to the flexura hepatica ; it is set up at the former point by gall-stones, neoplasms, etc., or is the continuation of the cirrhotic process in the liver or of a portal periphlebitis. In the left hypochondrium we sometimes find, together with numerous splenic adhesions and fibrous perisplenitis, the splenic flexure adherent and constricted by chronic fibrous peritonitis. Such contractions of the hepatic flexure of the colon are sometimes found in women whose livers have become grooved by lacing, in which case the fibrous perihepatitis is the starting-point of the latent peritonitis extending to the flexure of the colon.

A few weeks ago I saw another case of exquisite "laced liver" with severe habitual constipation, frequent pain in the right hypochondrium, occasional meteorism, especially in the region of the cæcum, and a fæcal tumor along the ascending colon which went under the distorted lobe of the liver. In 1871 a similar case was sent to the Clinic with the diagnosis of probable carcinoma of the gall-bladder. The prolonged use of enemata, together with mild laxatives, at length removed entirely the fæcal tumor, which had been mistaken for a malignant one, and thus allowed the grooved lobe to be recognized, under the sharp edge of which the fingers could be easily passed.

We also meet with the insidious process of chronic peritonitis in a diffuse form, spread over the greater portion of the peritoneum, especially of that covering the mesentery, and then it often presents a certain independent character, and causes thickening and shortening of the mesentery, thus binding the convolutions of the small intestine down to the vertebral column. This cirrhosis of the peritoneum ("peritonitis deformans," Klebs) results from chronic venous congestion in diseases of the heart, and sometimes exquisite examples are found with cirrhosis of the liver and atrophied nutmeg liver, and also occasionally with granular atrophy of the kidneys. We have already (p. 561) described in detail how chronic mesenterial peritonitis of the S loop may give rise to abnormal approximation and narrowing of its two ends and consequently to twisting of the sigmoid flexure.

Constriction of the Intestines by Neoplasms.

We have seen in the foregoing how new growths starting from the organs or walls of the abdominal or pelvic cavities may produce contraction by compression, or by peritonitic adhesion and resulting constriction, or by growth into the intestinal canal. We shall now consider *the neoplasms arising from the intestinal wall itself*; and of these we can distinguish two groups, differing essentially in their course, duration, and prognosis: *the benign and the malignant neoplasms*, or *cancers*. While the former usually project as polyps from a circumscribed point on the wall of the intes tine into its lumen and act as an obstruction (only seldom constricting it by exciting chronic peritonitis), the malignant growths or cancers act not only in the same way, but also and especially by constriction, since *they spread* rapidly around the intestine *in the form of a girdle* and cause notable *annular strictures*.

Among the benign neoplasms of the intestinal canal we distinguish:

1. *Fibromas* arising from the submucosa, which project, sometimes several at the same time, in the form of "fibrous polyps," into the interior of the canal and lead to obstruction or invagination.

2. *Myomas* arising from the muscular layer of the mucous membrane or from the muscular coat of the intestine, which are sometimes very vascular (angio-myomas), and sometimes fibrous (Myofibroids), and which as "polypoid myomas[1] may act like the preceding variety.

3. Submucous *lipomas*; these are pedunculated and project into the lumen of the intestine, are often multiple,[2] and but seldom become large enough to cause obstruction.

4. *Sarcomas*, in the form of tough polyps arising from the submucosa, are usually spindle-cell or fibrous sarcomas, very seldom the round-cell sarcoma, which is almost never in the form of a polyp, but spreads out like a belt in the submucosa and causes stenosis. Metastatic alveolar sarcoma, which is not unfrequently pigmented, and is then called melanosarcoma, pigment cancer (sarcoma melanodes, S. carcinomatos. pigmentatum), belongs to the malignant neoplasms.

5. The benign neoplasms arising from the mucous membrane appear in two forms, which are often combined: *a*, as *papillary polyps of the mucous membrane* (papillomas of the mucous membrane, papillary fibromas, benign villous polyps), due to hypertrophy and hyperplasia of the pre-existing villosities, and in the lowest part of the rectum to overgrowth of the papillary body of the mucous membrane;

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[1] *Böttcher*, Wagner's Arch. XI. 1870.

[2] *Huss*, Schmidt's Jahrb. Bd. 51, S. 171.

b, as *glandular polyps* or *polypoid adenomas*, consisting in hypertrophy and ectasia of the glands of Lieberkühn. They occur with relative frequency in the rectum in children, sometimes scattered in great numbers over the mucous membrane of the entire colon.[1] The so-called "flat adenoma" found in the rectum, which usually surrounds the canal in a ring and may give rise to constriction, belongs here as a pure adenomatous form. Hypertrophy and ectasia of the glands is much more frequently associated with papillary overgrowth in the formation of polypoid tumors, of the "adenoma or fibro-adenoma papillare" (Klebs). But here we meet with transformation into epithelial carcinoma. Papillary adenoma may be caused by the irritation of a neighboring carcinoma, which, by infiltration from below between glandular tubes and into the papillæ, changes the apparently benign papillary adenoma into the real, destructive villous cancer; or the originally benign papillary adenoma extends more deeply, oversteps the boundary between epithelium and connective tissue, breaks down, and becomes carcinoma, transformations which often take place in the single, broadly implanted adenomas and papillomas of the mucous membrane, but almost never in those that are pedunculated and multiple.

The polypoid neoplasms above mentioned usually give rise to no symptoms unless they reach a certain size, or, when located in the rectum, cause tenesmus, hemorrhage, and difficulty in defecation. Except lipomas and myomas, they are usually situated in the rectum. They are, however, found also in the colon, cæcum, at the ileocæcal valve, in the upper and lower portions of the small intestine, in the duodenum, and most rarely in the middle portion of the ileum.[2] They sometimes give rise to acute occlusion of the intestine[3] by congestive swelling and rapid growth following many indications of trouble in the abdomen, the cause of which remains obscure, or to invagination, espe-

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[1] *Luschka*, Virch. Arch. XX. 133. *Reinhardt*, Charité Annal. Heft I. 1851. *Boyer*, Prag. Viertelj. 1844. *Billroth*, Langenbeck's Arch. 1869. *Sibley*, Lond. Path. Soc. VII. 212. *Hulke*, ibid. XIV. 173.

[2] In my extended examination of the literature of this subject I noted the facts concerning intestinal polyps also. Their situation was as follows:

Rectum	75 (or more).
Different places in the colon	10
Cæcum	4
Ileocæcal valve	2
Lowest part of the ileum	30 (most of them invaginations).
Jejunum	5
Duodenum	2
Total,	128

[3] *Croft*, Lond. Path. Soc. XIV. 155.

cially in the lower part of the ileum. The diagnosis can be made only when the polyp is situated in the rectum or in the rare cases when it comes away.[1]

Carcinoma of the Intestinal Canal.

Cancers appear primarily and secondarily in the intestines. The latter is by metastasis or by extension to the intestine of a carcinoma of the neighboring parts. We distinguish between the *belt-shaped cancer*, which usually produces constriction promptly, and *diffuse cancerous infiltration* of the intestinal walls. The latter often causes only an inconsiderable constriction of the canal, but it leads to arrest of the contents, and in this way to occlusion of the intestines, because it renders a portion of the intestine incapable of peristaltic action.

Among primary cancers of the intestine we include:

1. *Scirrhus* (carcinoma fibrosum); this usually forms very tough and narrow strictures.

2. *Soft carcinoma* (C. medullare, encephaloid); this forms large, belt-like tumors, usually undergoing necrobiosis promptly and becoming transformed into ulcers. This cancer differs from the preceding one in being vascular, and gives rise to hemorrhages. It is also peculiarly true of this that, after it has long been the cause of a narrow stenosis, the canal often becomes free as the result of its destruction and ulceration. It gives rise to early cachexia, extends rapidly, not unfrequently causes perforation, decomposition of the cellular tissue, etc. In other cases the callous thickened base, the cancer star, which is enclosed on all sides by a carcinomatous wall, causes tough fibrous stricture (fibromedullary carcinoma, "constricting form of medullary cancer" (Engel).

3. *Colloid cancer* (C. alveolare, colloides); this forms large tumors and acts by obstructing, but mainly by constricting.

4. *Cylindrical epithelial cancer* (identical with the "destructive papilloma of the mucous membrane, adenoma carcinomatosum, medullary villous cancer, destructive adenoma"); this has its origin in the glandular cells of the Lieberkühn crypts, with participation of the epithelium of the papillæ; it is therefore a real glandular cancer.

5. *Sarcoma alveolare* and pigment cancer.[2]

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[1] *Castellain*, Gaz. hebdom. 1870, No. 20. *Portal*, quoted by Duchaussoy, l. c. *Jetter*, Württemb. Corresp.-Blatt, 1872.

[2] Carcinoma of the intestine is situated, by far the most frequently (in 80 per cent.), in the rectum. As for the statistical relations—for which the reports of the k. k. allgem. Krankenhaus at Vienna are most suitable—a careful collation gives the fol-

Symptoms, Diagnosis.

The symptoms and course of cancer of the intestine vary according to the location and the anatomical kind and shape of the carcinoma. We can, in general, divide them into two frequently occurring kinds: first, those in which the first symptom

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lowing results: Out of 34,523 deaths at the hospital just mentioned, between the years 1858 and 1870, were 1,874 cancers of different kinds, 5.4 per cent., a number with which the quite reliable Geneva list agrees, although the many smaller statistics do not. Of 4,567 cancers at the same hospital 143 were of the rectum, and 35 of other parts of the intestines; the former were, therefore, 3 per cent., the latter 0.76 per cent. of the whole; the former 80 per cent., the latter 20 per cent. of all cancers of the intestines. The Geneva list and the London statistics (Med. Chir. Transact. T. 42, 1859) agree entirely with reference to the proportion of cancer of the rectum, but they give a higher figure (2 to 3 per cent.) for cancer of other parts of the intestines, because cancer of the peritoneum is not separated as distinctly from that of the intestine as is the case in the Vienna statistics. Statistics made up from published cases furnish useless and partly contradictory figures. But still it is not only allowable, but necessary to use published cases in determining the relative frequency of the different seats of cancer of the intestine, all the more so because these are much more accurately described than is the case in most hospital reports and mortuary records. The published cases collected by me, together with the records of pathological institutes and hospitals, give the following trustworthy figures:

Cancer of the colon (exclusive of rectum)	89	cases.	Sigmoid flexure	42
			Descend. colon	11
			Transverse colon, including flexures	30
			Ascending colon	6
Cancer of cæcum	20	"		
" " Appendix vermiformis	3	"		
" " ileocæcal valve	9	"		
" " small intestine	33	"	Duodenum and jejunum	17
			Middle portion of ileum	3
			Lower " "	13
Total	154			

These 154 cases of cancer of small intestine and colon correspond (according to the foregoing) to 616 cases of cancer of the rectum, and we obtain, by a further calculation, the following proportions for cancer of the intestines in general: cancer of the rectum 80 per cent., of the colon 11.5 per cent., of the cæcum (including the ileocæcal valve and vermiform appendix) 4.1 per cent., of the small intestine 4.3 per cent. Opinions regarding the relative frequency of cancer of the intestines in the two sexes differ greatly. Thus the proportion of males to females is placed by Desault at 1 to 10 (!), by Billroth at 10 to 8; Bérard, 20 to 23; Rokitansky, 15 to 17. Of the 143 cases of carcinoma of the intestine, in the above statistics, 80 were men and 63 women. Of the other 35 cancers of the intestines, 19 were in men, 16 in women. Before the age of 40 years, cancer of the intestine is very rare; it is most frequent after the age of 50. Exceptionally, cases occur in youth; thus, in a child three years old (Clar), in one of 12 years (Majo), one of 16 (Busk), one of 21 (Bruch). Duchaussoy's statistics give an average duration of 8 months for different cancers of the intestine; that of cancer of the rectum, according to Billroth, is 16 months.

is a disturbance of general nutrition, emaciation, change of physiognomy, of the color of the skin, of the voice, in short, the signs of a process affecting nutrition which cannot be localized at first. Then follow, gradually, symptoms which point to an affection of the abdomen, at first still obscure ; then a hardness, a painful tumor is discovered in some part of the abdomen ; and finally are superadded the symptoms of obstruction of the advance of the contents of the intestine. In other cases, especially in cancer of the rectum, the symptoms at the beginning are purely local, apparently unimportant ; the patients think they are suffering from hemorrhoids, which are not unfrequently present at the same time. The physician, too, may be in doubt at this moment whether the hardness, the tumor which he feels per anum, is of malignant origin or an inflammatory infiltration of the mucous membrane, caused by chronic catarrh, or a benign papilloma of the mucous membrane, etc. For a long time the body remains fat and the appearance healthy, but at last, with the growth or the breaking down of the neoplasm, the indications of cancerous marasmus appear.

Cancer of the intestines commonly causes violent, lancinating pains in the abdomen, often radiating to different parts of the body. When it is situated in the rectum and colon, pain in the sacrum and back especially are present. An inclination towards constipation usually appears early, and may become exceedingly obstinate as the constriction increases ; or constipation and diarrhœa lasting for several days may alternate irregularly. We are often astonished to find at the autopsy very tight stenoses when there had been no marked obstruction of the fæces during life, but, on the contrary, constant diarrhœa. The products of the severe intestinal catarrh, together with the irritation exciting peristaltic action, which the contents of the intestine produce in the cancerous ulcer, never allow the fæces to thicken, but hasten its passage, and thus overbalance the effect of the stenosis. When the cancerous mass breaks down, the evacuations present a very offensively smelling, ichorous condition, and are mixed with blood. Severe hemorrhages are rare. They are found chiefly with villous cancer. The deeper the seat of the cancer in the colon or rectum, the greater and more general the meteorism, the more severe the tenesmus and the pain at every defecation. The detection of a tumor in the abdomen is of the greatest importance in the diagnosis of cancer of the intestines. It is usually hard and irregularly knobbed. On account of the greater frequency of cancer of the colon and cæcum, the tumor is usually found at a point corresponding to the course of the colon or in the region of the cæcum, where it may be confounded with typhlitis and perityphlitis.[1] Still it is to be remembered that cancerous tumors of the transverse colon, ileum, and sigmoid flexure often sink downwards and can be felt in the hypogastrium, sometimes in the true pelvis through the rectum or vagina. That displacements of this kind may, by the production of bands and adhesions, create new and notable obstacles to the advance of the fæces is evident. The hypertrophied walls of the intestines above the cancer often show their outlines in relief through the emaciated walls of the abdomen, and can sometimes be felt as stiff tubes.

Diagnosis of cancer of the rectum is the easiest, for at its usual seat, two or three inches above the anus, and even when higher, it can be touched. When the carcinoma is situated in the duodenum, the symptoms—frequent vomiting, sometimes even of blood, retracted abdomen, ectasia of the stomach, obstinate constipation, "cardialgia," tumor in the epigastrium—often resemble cancer of the pylorus so closely that mistakes can scarcely be avoided. The fatal termination in intestinal cancer is due either to occlusion of the intestine with symptoms of ileus, or to profound cancerous marasmus, often with spread of the carcinoma to the adjoining organs, or to perforation, peritonitis, or decomposition of the retroperitoneal cellular tissue, with consuming fever and metastases of the cancerous and putrid matter. The diagnosis of the rare pigment cancer is often made possible at an early period by the presence of pigment cancers in the external skin. Besides the above-described symptoms, the hereditary predisposition, the age of the patient, and the previous duration of the disease, must be considered in making the diagnosis.

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[1] *Bamberger*, Oesterr. Zeitsch. f. prakt. Heilk. III. 8. 9. 1857.

Statistics of Occlusions and Constrictions of the Intestines.

The question of the frequency of the occurrence of these affections can be answered but incompletely in the present condition of the lists of patients and deaths. The different kinds of occlusion of the intestines are partly given as "ileus, volvulus, internal strangulation," and partly concealed under the title of "inflammation of the peritoneum." The error resulting from the latter circumstance is compensated for, and perhaps overbalanced, by the great number of those cases which are improperly entered with the diagnosis of "ileus," a term about the use and extent of which physicians still differ. But there are many other circumstances which give statistics like those presented at least the value of an approximative probability. Without wishing in the least to question the great value of those statistical figures which are based upon the sanitary records of a population the size of which is established by census, I must still say, with reference to the statistics of occlusion of the intestines, that the records of large general hospitals, extending over many years, are of greater statistical value for our purpose, since they have the advantage of a strict discrimination of the individual cases of intestinal occlusion, not only in the diagnosis, but also and especially by means of the autopsy. In the following we compare the figures obtained in both ways, and thereby limit ourselves to only a few opinions; this is not the place nor have we room enough for statistical details.

The mortuary records of England, covering a period of six years, show that of 100,000 inhabitants nine die every year of constriction or occlusion of the intestines (exclusive of external hernias and malignant neoplasms); the records of the Canton of Geneva show that of 100,000 inhabitants five die every year from the same causes.[1] I have collected for comparison with these figures the statistics of the k. k. allgem. Krankenhaus in Vienna,[2] from 1858 to 1870, and also those of different hospitals in St. Petersburg[3] from 1867 to 1871, and have calculated from the mortality records the number of inhabitants corresponding to the number of patients in these hospitals (a total of 425,918 patients). The result showed that in Vienna six, in St. Petersburg ten, of every 100,000 inhabitants died from constriction or occlusion of the intestinal canal; and we may therefore say that, generally speaking, from five to ten fatal cases of occlusion of the intestines occur yearly among every 100,000 inhabitants.

According to the English records for the period of time mentioned, of every 260 deaths one was due to occlusion and constriction of the intestine (external hernias and malignant tumors always excluded, as in the following also). In a total of 12,000 autopsies Brinton found death caused in one case out of every 280 by constriction or occlusion of the intestines, a number which agrees very well with the preceding. On the other hand, in Geneva, during a period of thirteen years, the proportion of deaths by occlusion of the intestines was one in 416; of 34,523 deaths at the general hospital in Vienna, sixty-eight were due to occlusion of the intestines, or one in 500; at the St. Petersburg hospital, one in 330 (25,396 deaths with seventy-five by occlusion); the anatomico-pathological institute at Prague [1] from 1845 to 1873 (with interruptions) gives a proportion of one to 240 (13,105 autopsies with forty-five occlusions of the intestines). Considerable differences are found here also, and we shall best express ourselves by saying that there is one case of constriction or occlusion of the intestines in every 300 to 500 deaths.

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[1] *Oesterlen*, Handb. d. med. Stat. Tüb. 1865, S. 638, S. 749, et seq.

[2] Aerztl. Bericht d. k. k. allg. Krankenh. in Wien.

[3] Collected from the Petersb. med. Ztg.

As for the influence of sex and age, we have already given the most important points, when speaking of the different kinds of occlusion and constriction. The different statistics differ widely upon these points. If we consider first the proportion of males and females affected by the different kinds of occlusion, the published cases collected by me give the following figures

Strangulation by false ligaments	52	males.	59	females.
“ by the omentum	43	“	15	“
“ by diverticles	52	“	14	“
“ by the appendix vermiformis	21	“	13	“
“ in internal hernias	25	“	6	“
Diaphragmatic hernias	163	“	52	“
Intussusception	285	“	157	“
Obstruction by gall-stones	9	“	32	“
“ by foreign bodies	37	“	10	“
“ by intestinal stones	15	“	5	“
Strangulation in holes and fissures of the mesentery, in abnormal openings in different organs and parts of organs, in rings formed by the adhesion of abdominal or pelvic viscera with each other or with the walls	12	“	17	“
Compression of the intestine by the mesentery	10	“	8	“
Compression of the intestine by tumors, viscera, etc	15	“	37	“
Twisting of the sigmoid flexure (and ileum)	23	“	10	“
Knotting of two intestinal loops	20	“	1	“
Acute bending by displacement (with or without simultaneous compression by the mesentery)	8	“	6	“
Ileus paralyticus, fæcal obstruction	10	“	15	“
Total	800	males.	457	females.

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[1] Collected from the Prager Vierteljahrschrift.

Constrictions of the intestines (whether they caused death by ileus or otherwise), chronic kinks, different kinds of stricture, constriction by chronic peritonitis, adhesions, etc.	35 males.	71 females.
Cancer of the intestine	19 "	16 "
" " rectum	80 "	63 "
Grand total	934 males.	607 females.

Examination of these tables shows that males are more often affected than females by the majority of the causes of occlusion of the intestines; the exceptions are chiefly compression by tumors, obstruction by gall-stones, and constriction by partial chronic peritonitis. But it would be very erroneous to infer from the total given above, that the proportion of males to females in these affections taken together is as nine to six. This question cannot be answered by the collation of published cases, but only from the mortality records of a community, or, if need be, from the records, covering many years, of large hospitals, for the relative frequency of the different kinds of occlusion is very different from that given above. It happens only too frequently that senseless statistical conclusions are drawn from collections of published cases. The English tables of mortality, for the period above mentioned, show an unimportant excess of males over females (8.6 to 8.3); the Geneva records show even a slight superiority of the latter (22 f. to 18 m.); the general hospital at Vienna shows the proportion of males to females, between the years 1858 and 1870, to have been as 5 to 4 for occlusion, and 2 to 3 for constriction of the intestines.

As for the relative frequency of the different kinds of occlusion and constriction, as compared with one another, we possess no sufficient statistics. The large mortality tables are of no use here, for they distinguish the cases of intussusception only; and the records of large hospitals, also, as I found after much trouble,

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[1] Thus those who died in England, in the year 1859, of occlusion or constriction of the intestine may be divided, according to age, as follows. In every one hundred cases there died of:

Aged from	1. "*Ileus and Volvulus.*"	2. *Stenosis.*	3. *Intussusception.*
0 to 1 year	15.4	1.7	19
1 " 5 "	5.0		5.2
5 " 10 "	4	0.3	5.2
10 " 15 "	4.2	0.3	6
15 " 25 "	7.3	2.8	8.1
25 " 35 "	5.6	7	6
35 " 45 "	9.6	10.7	10.3
45 " 55 "	10	21.1	9.2
55 " 65 "	14	24.6	1.5
65 " 75 "	16.3	25.6	.5
75 " 85 "	7.6	3.8	4
85 " ——	1	2.1	0.7

allow, in a certain sense, only approximative opinions. One might be tempted to make a collation of a great number of published cases, somewhat like that given above, so as to determine this question. I shall protect myself from this error, which is still committed daily, for this method would yield estimates far exceeding the truth for the rarer kinds of occlusion, and vice versa.

As for the age, the English mortality records give too high figures for early childhood (up to the fifth year),[1] because the diagnosis "ileus" is very widely and variably used at this age, as has been already said. If, however, the curve of intestinal occlusions, arranged with reference to the age, shows two elevations, the first in the first year of life, and the second between the forty-fifth and sixty-fifth years, the former is due chiefly to the relative frequency of invagination in childhood, especially during the first year.

As for the percentage of recoveries from ileus, I have at my command no other information than that contained in the statistical reports of the general hospital at Vienna—namely, that out of sixty cases of ileus six, or ten per cent. recovered.

Treatment.

If, notwithstanding the variety of the anatomical processes which occasion occlusion and constriction of the intestines, I consider the treatment of them all together, there are practical considerations which induce me to do so. How often is it impossible for the physician, at the bedside of a patient presenting the severest symptoms of internal strangulation, to form an opinion as to the special anatomical cause of the impermeability of the intestine, notwithstanding the most thorough examination and most careful consideration of all objective and commemorative indications. Not unfrequently this inability to form a definite anatomical diagnosis is the reason why the treatment of occlusion of the intestine remains so impotent. But our treatment—operative interference excepted—would not be different even if we were able to say whether the cause of the occlusion was a strangulating false ligament, a diverticle, or the appendix vermiformis; whether a fissure in the mesentery or omentum, torsion, knotting, or acute bending; whether the lodging of a gallstone in the ileum, compression by the mesentery, or an acute invagination of the ileum. On the other hand, those causes of occlusion, diagnosis of which during life may be important in indicating the choice of special therapeutical measures, can actually be recognized in the great number of cases. Among these are occlusions of the rectum, accessible by direct treatment, fæcal obstruction of the colon, ileocæcal and colon invaginations, compression by tumors, cysts, etc. In the greater number of all other cases of acute impermeability the knowledge of the anatomical nature of the obstruction certainly has a bearing upon the result of operative interference; but so far as other treatment is concerned, it is of subordinate importance.

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[1] See foot-note at bottom of preceding page.

The history of the treatment of ileus gives us a true reflection of the ruling opinions concerning the nature of this affection. From the times of Hippocrates, Aretæus, and Galen, who considered ileus an inflammation, and therefore recommended venesection and "cucurbitulas pluribus locis admotas" (Celsus), until a few decades ago, the treatment by letting blood once, or several times, was almost universally employed. After the sixteenth century physicians felt the more justified in employing it, because an inflammatory condition of the intestines was always found at the autopises. Besides venesection, the most varied rules were laid down; hot fomentations over the entire trunk [1] (Celsus), cooling of the arms and feet, friction applied to the extremities, placing the whole body in a bath of warm oil, and internally aperients [2] (warm oil, turpentine, "nitrum," honey, etc.), or carminatives (anis, fennel, petroselinum) were ordered. Furthermore, enemata and inflation of air per anum, as well as narcotics [3] were important links in the chain of therapeutical measures. Besides the treatment directed against the inflammation, to which was afterwards added the use of large blisters, calomel pushed to salivation, friction with mercurial ointment, etc., measures were naturally taken to overcome the constipation. The long line of purgatives, from the mildest laxatives to gamboge, scammony, colocynth, and croton oil, was brought into action, blow upon blow. They did not try, in ancient times, only to overcome the constipation as such; their intention was also to remove the cause of the ileus, the "humores acres intra intestinorum membranas inhærentes," the "acrimonias arthriticas, biliosas, rheumaticas," the "excandescentiam spirituum animalium," the "humorum anarrhopiam," and to rectify the "motus intestinorum inversus." If the usual purgatives did not succeed, they had recourse to still other drastic measures. They were not afraid to administer enemata of infusion of cantharides (Wilmer) with a solutio tartari emetici (Quarin), or to assist vomiting in order to "relieve the conamen naturæ of sharpness," for they gave emetics "continuously" (Nicol. Piso). Seldom was a voice (Massaria)[1] raised against these crazy attempts to purge.

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[1] Partly to alleviate pain, partly with the view of making the skin thinner, and favoring the escape of gas through it; for the "trajectio spiritus" was considered the cause of ileus. (Hippocrates, De flatibus. Edit. Kühn, T. I. 578.)

[2] While Hippocrates (De Morb. Lib. III. p. 304) placed "Venter quam citissime purgandus" first, Celsus (Lib. IV. C. 13) made this distinction: "Si supra umbilicum malum est (morbus ileus) alvi ductio utilis non est; si infra est (morbus colicus), alvum ducere optimum."

[3] "Siquidem hominibus ita dolore cruciatis vel mori beatum est, mortem tamen cuivis inferre medico nulli nobili fas est; fas autem aliquando, cum præsentia mala evitari non posse manifeste prævideat, ægrum sopire." *Aretæus*, Lib. II. Ed. Kühn, p. 272.

With the theory of the nervous, especially spastic nature of ileus, the antispasmodics came into favor, by which opium was much more rarely meant than belladonna, nicotiana, cicuta, and hyoscyamus, extensive use of oil internally and externally, warm baths, and the like; besides this, regular venesection, to produce "relaxation of the contracted intestine." Finally, there were not lacking other recommendations, often of the most singular kind; for example: "Cutis calida a vivente agno detracta abdomini applicabatur;"[2] or Sydenham: "Catulum viventem nudo ventri indesinenter accumbere jussit." Others claimed specifics—for example, the expressed juice of six pounds of decaying Borsdorf apples.[3]

As with the advance in pathological anatomy it became more and more clear that the existence of mechanical obstructions was a cause of ileus, and mechanical ileus took the place of the dynamic ileus which had previously been almost exclusively accepted, measures were recommended which should forcibly break through the obstructions. Diocles, the Carystian, had used balls of lead, without, however, finding favor. Henricus Screta was, as Peyer states, one of the first to administer "globulos ex electro metallico antihectico Poterii," balls of antimony and tin. His example found eager imitation. Pounds of shot, balls made of metallic antimony, and other things were now the favorites. Sylvius de le Boë[4] went to the luxury of gold and silver balls, although, as one of his contemporaries assures us, only that, at a time when autopsies were not in favor, he might be more sure of permission to open the body after the death which he saw to be certain. Van Helmont went so far in his enthusiasm over this method of treatment as to declare: "Neminem ileo perire si globuli plumbei musquetarum deglutiantur."[5] All these mechanical means were again laid aside when a substitute was found in mercurius vivus, which was not only more agreeable to swallow, but also exerted a uniform and considerable pressure. The use of this remedy soon became so widespread, that, as Rosinus Lentilius assures us, it was seldom a patient died without having taken metallic mercury. Zacutus, the Lusitanian,[6] Marianus Sanctus, and others gave three pounds of mercury, and the patient was at the same time rolled, shaken, or, as I read in Hoffmann,[1] made to walk up and down the room. A. Paré,[2] Rhodius,[3] De Haën,[4] van Swieten (l. c.), and many others praised the remedy,[5] which was then used also for simple constipation, and enjoyed such favor that, at least according to van Swieten, it was the fashion at that time in England to take every morning one or two ounces of quicksilver—a treatment which never presented any disadvantages. Metallic mercury as a remedy in ileus has had its adherents even until our times; but, fortunately, they have been steadily growing fewer. Under the title, "Machinæ ad ileum curandum casu inventæ descriptio et usus, Mailand, 1765," Videmar[6] finally offered an instrument for administering enormous injections, which had also been recommended by Mollinari, De Haën, and others, in ileus.

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[1] Op. med. Lugd. 1634.

[2] Med. Essays, T. V. Pars. 2, p. 893. Others praised cold very particularly as a cure. The patient was made to drink freely of cold water. (*Hoffmann*, *Amatus Lusitanus*, *P. Forestus* (Lib. 21. Schol. ad Observ. 12,) *J. Crato* (Lib. III. Cons. 13)). *Ballonius* placed large blisters upon the abdomen.

[3] *Winkler*, in *Bonet*, Med. Sept. Lib. 3, C. 30.

[4] Idea prax. med. Lib. I. C. 15, § 53.

[5] De flatibus, § 31.

[6] De med. Princ. hist. Lib. II.

If the physician at the bedside has convinced himself of the existence of an internal strangulation; if careful examination of all the hernial openings, including the obturator foramen, has shown the absence of strangulated external hernia; if the rectum is found to be free; if examination of the vagina gives no indication of the cause of the strangulation; if full enemata and the exploration of the abdomen prove that there is no fæcal accumulation, and that the lower part of the colon is not the seat of the strangulation—in the latter case the usual kind of knotting and twisting is excluded—then treatment with *opium* is to be begun. The more acute the symptoms of impermeability, the more intense the colics, and the more severe the whole train of symptoms from the beginning, so much the more clearly is opium indicated. It is very probable that the violent peristaltic movements excited by the reflex action of the local injury caused by the strangulation, the existence of which can be easily ascertained by laying the ear directly upon the abdomen, can, as a rule, only be disadvantageous. They certainly increase the strangulation often, for they narrow the strangulating ring, or bring fresh portions of the intestine into it; by their constant pulling at the point of strangulation they occasion earlier inflammatory infiltration, swelling, and exudation, and thus promote fixation of the strangulation; they indirectly favor venous congestion of the strangulated loop, and thereby prepare the way for gangrene and total paralysis; finally, they aid in exciting, not only a local adhesive, but also a general peritonitis, and greatly increase the pain. Is it rational in such cases to intensify the local injuries and add fresh ones, to try to increase the already too great peristaltic action, with its damages and dangers, as unfortunately still happens too often, by the administration of purgatives, and usually, indeed, not the mildest? While the surgeons have long since repudiated the use of purgatives in strangulated hernia,[1] this mode of treatment of internal strangulation still counts numerous adherents in the professorial chair as well as in practice, and the principle laid down by Stoll (T. I. p. 249) for the treatment of ileus—"non esse a medico nocendum, ubi is prodesse nequeat"—is greatly sinned against. Many, indeed, hope in cases of internal strangulation to break forcibly through the obstruction by means of drastic purges, and they do not shrink from the accompanying dangers. Though such a procedure may have the wished-for effect in some rare cases, I still believe that it is more often injurious than useful. I think that, as in the disentanglement of a knot, gentle efforts are more successful than simple force applied unintelligently, so, too, in internal strangulation the quiet produced by opium, and perhaps even the correction of the peristaltic action, are far more useful than an irregular excess of the latter. Since opium stops the vomiting, it favors the filling with liquid contents of the portion of the intestine which lies above the obstruction, and thus, associated with regular peristaltic action, may aid in the removal of the latter. Beyond any question, opium is an important means of preserving the intestine, and often life.

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[1] Med. rat. T. IV. P. 2, p. 335.

[2] Op. chir. Lib. XX. C. 38.

[3] Obs. med. Cent. II. Obs. 80.

[4] P. XI. C. III. p. 189, et seq.

[5] The possibility of perforation of the intestine was never thought of. Only a single writer, Rich. Mead, 1744, mentioned the danger of rupture of the intestines. The only fear was that the quicksilver might cause injury by cold (!) (Henric. ab Heer, Lib. I. Obs. 2).

[6] See De Haën, Rat. med. T. VIII. C. V. p. 241: "Machinæ quæ uno tenore 6–8 libras injicit, ad ileum curandum casu inventæ descriptio et usus." Mailand, 1765.

If the opium is to overcome the above-mentioned dangers, and quiet the vomiting, jactitation, and pain, it must be given to adults, with whom, almost exclusively, we have to deal, in energetic and rapidly repeated doses. Observation of the patient furnishes the measure of the amount. It must be given until slight narcosis begins, until the vomiting stops. If the latter prevents the taking of the opium by the stomach, the same result may be obtained by small enemata containing it, or by repeated small subcutaneous injections of morphine. It has been urged against opium that it increases the usually existing collapse, and is dangerous in this way. Without denying that there is a moment in the treatment of ileus when all the efforts should be directed simply and solely to maintaining the endangered life by means of the most powerful stimulants, yet, on the other hand, when I was the pupil and assistant of Pfeufer, who was one of the first in Germany to strenuously recommend[1] and to practise with great consistency at the bedside the energetic treatment with opium, from the beginning, of all kinds of internal strangulation, as well as peritonitis and typhlitis, I repeatedly saw that when the influence of the opium began to be felt, as the patient grew quiet and the severe vomiting ceased, the collapse also diminished, the turgescence of the skin returned (the result, I suppose, of the renewal of absorption of water by the blood), the pulse and the lowered temperature of the body increased, the leathery dry tongue grew moist, the facies of the patient improved, and he again became able to take food and stimulants, and to recover his self-control.

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[1] Hear, for example, what Rust says: "The use of purgatives and irritating enemata in this kind of strangulation is an offence, not only against all the principles of advanced medical science, but also against common sense; it only makes the progress more acute."

In addition to the opium, *cracked ice* should be given to relieve the tormenting thirst and the vomiting. Black coffee is recommended by many with the same object. The application of compresses or bags of ice upon the abdomen seems to me to be allowable only after the intestines have been quieted by large doses of opium. If their effect is to excite peristaltic action, as is shown by the recurrence of pain and can be demonstrated by auscultation, they must be removed. On the other hand, they promise, like moderate local abstractions of blood, to render useful services against the existing or threatening general peritonitis. Priessnitz's compresses often contribute essentially to alleviate the pain. General bleeding is to be rejected on principle. Little can be expected from the use of mercurial ointments and plasters, from blisters and dry cups.

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[1] Zeitschr. f. rat. Med. N. F. II. 1. 1851. Pfeufer's proposition at first received but little notice, and his practice found imitators only among his pupils. Wachsmuth's warm recommendations and clear explanations (Virch. Arch. Bd. 23, 1862, S. 144) first brought the opium treatment of ileus into extensive use in Germany also. In England it had long been used and prominently recommended. For example, Habershon (l. c., p. 475) thus expresses himself: "Purgatives of all kinds are better avoided, and the use of drastic measures will tend to aggravate the sufferings, to shorten life, and remove the possible chances of recovery. The administration of opium is now known to be attended with beneficial results, and frequently with partial, if not with permanent relief. By this means the peristaltic action is checked, spasmodic contraction diminished, and the opening, which previously would not allow the passage of flatus, will suffer fluid fæces to escape."

Even if the opium treatment in ileus is far superior to all others, it is not to be undertaken blindly. We can distinguish two varieties of occlusion of the intestine, according to the mode of appearance. The first is the acute one, the patient being suddenly attacked by exceedingly violent colicky pains. In such cases opium is at once indicated, whether there is constipation or not, whether it is a severe fæcal colic or the beginning of a typhlitis or strangulation of the intestine. Cannot the colon or rectum be emptied by injections as well or even better under the quieting influence of opium? I remember cases of typhlitis in which the patients had been constipated for several days before the appearance of violent colics. Exclusive opium treatment was at once begun and carried on until the pain had disappeared entirely, and then spontaneous evacuation of the bowels followed several days afterwards. I remember that in such cases Pfeufer administered enemata only if the patient had no spontaneous pain and no sensibility to pressure on the abdomen after the opium treatment had been suspended twenty-four hours. In other cases, the appearance of acute occlusion of the intestines is preceded for several days by the symptoms of incomplete evacuation of the bowels, distention of the abdomen, slight colics. At this time, when there can be no suspicion of the coming occlusion, enemata, calomel, castor-oil, and other mild purges, are always used, and indeed often with advantage to the patient, until, with the appearance of most intense colicky pains, vomiting, and commencing collapse, the diagnosis of occlusion becomes possible. If all the signs of existing constriction have been present for a long time, and if the symptoms grow more severe, if the stools occur at longer intervals than usual, if meteorism appears, in short, the symptoms of increasing obstruction of the passage and stagnation of the contents, then the administration of a few doses of castor-oil or calomel, together with enemata of cold water, may prevent the threatening impermeability. If, under these circumstances, the physician has to deal with a chronic retardation of the fæcal current in the colon, caused by primary atony or by its abnormal disposition, or with fæcal obstruction of the rectal ampulla, the repeated and persistent use of large enemata of warm water, best employed according to Hegar's method (vide supra), or, if necessary, the direct removal of the hard scybala with the fingers or instruments seems to be indicated. At the same time the softening and breaking up of the fæcal masses are aided from above by purgatives. But in all these cases, if intense colicky pains, rapidly increasing meteorism, vomiting, sensibility of the abdomen to pressure, in short, the symptoms of severer occlusion or peritonitis appear, the opium treatment is to be instituted, and, after its soothing effect has been obtained, the gradual removal of the fæcal masses in the colon can be accomplished by repeated enemata.

So long as we have in opium a remedy certain in its effect and dose, it is inconceivable why any one should substitute for it belladonna, which varies so much in its preparations, doses, and effects upon different people, is so uncertain, and so often occasions unpleasant symptoms of poisoning, or cicuta, or hyoscyamus, remedies of which we are not even sure how they act upon the intestines. While, for example, von Bezold and Bloebaum maintain that belladonna arrests peristaltic action, Keuchel saw no change produced by it, and others infer from its effect in favoring the passages that on the contrary it excites peristaltic action (Trousseau). Enemata containing an infusion of tobacco, more rarely insufflation of tobacco smoke, have enjoyed great favor and received many commendations even up to the present time. The expectation that the nicotine would quiet the peristaltic action and the "spasmodic contractions" has been proved to be illusory by recent investigations, which showed that on the contrary peristaltic action was excited and made more vigorous by this drug, and that paralyzing doses of nicotine carried with them the danger of most unpleasant poisonous effects.

If, notwithstanding the use of opium, the symptoms of impermeability continue, if the collapse increases and the hope of spontaneous removal of the obstruction grows steadily less, it is still recommended in such "doubtful cases" as a "last resort" to give metallic mercury in doses of from one hundred to three hundred grammes until from one to two pounds have been taken, in order to break through the obstruction and forcibly restore permeability. Like all other measures recommended in ileus, medical literature contains a series of successes, of "recoveries from ileus," attributed to metallic mercury also. These should not prevent us from criticizing this remedy briefly, the more so because there are still many erroneous notions concerning its mode of action and efficiency.

In the majority of cases metallic mercury is used late, after the ileus has lasted for some time. If, in such a case as we suppose, the whole of the metal makes its way to the point of strangulation, it is certainly far more probable that it will cause rupture there by its weight than that it will be able to remove the obstruction against which the most powerful peristaltic pressure has struggled in vain. If, nevertheless, the termination by rupture seldom happens after the use of mercury, the reason is that in most cases the metal does not get as far as the strangulated spot.

Ebers,[1] Hanius,[2] and Traube[3] have shown that it often remains in the stomach. This happens when the remedy, as is usually the case, is given at a late period. The already paretic stomach is paralyzed by the weight of the quicksilver; many observations show that immediately after it has been given the vomiting often ceases (J. Frank, l. c., S. 540, Hanius, Hauff[4]), and the paralysis of the stomach and intestines produces an ante-mortem condition of comfort, such as not unfrequently precedes death by occlusion of the intestines. In other cases, if the mercury is taken earlier, at a period when the stomach and intestines are still capable of peristaltic action, and the patient's condition is better, it makes its way, after a longer or shorter sojourn in the stomach, through the pylorus into the intestines. It is then divided up into a great number of small particles, which are found scattered here and there (Ebers[5]), and in some places collect together again in larger balls and masses. When these accumulations occur, peristaltic action, if the intestine is still excitable enough, is set up by the reflex action of the accompanying stretching of the intestinal membranes, active movements of the intestines take place, and the accumulated masses of mercury are again broken up into small particles and driven in different directions. It is very probable, as Hanius (l. c.) stated, and Traube afterwards insisted upon, that movements of the intestines may be excited in the same reflex manner by quicksilver lying in the stomach. This opinion is confirmed by the fact that mercury taken to relieve severe coprostasis often has a laxative effect in the course of a few hours, while it first appears in the stools several days, in Ebers's case nine days, afterwards. That the metal is divided up into small particles in the intestines, as above described, is proved, besides what is found at autopsies, by the fact that, in cases of occlusion that are recovered from, the quicksilver is not evacuated in a single mass with the first stools, but always very gradually in small pellets, which are often found mixed with the passages for weeks and even for months (Luzzato,[1] Hanius, l. c., Ficinus,[2] Fraentzel[3]). The progress of quicksilver then through the intestines is usually very slow. Even De Haën noticed that mercury given forty hours before death, "partim in ventriculo, partim in duodeno fuit,"[4] and in Pillore's patient four weeks after two pounds of metallic mercury had been administered, the whole amount was found in the convolutions of the jejunum. We may then say that in most cases of acute internal strangulation the recovery or fatal termination is decided before an efficient amount of mercury has been collected above the obstruction. But even in those cases in which life is prolonged for some time after the administration of quicksilver, and a sufficient amount of it has collected above the obstruction,[5] success is still doubtful. The metal may cause rupture or favor inflammation and gangrene, or make pressure at the wrong place, or cause paralysis of the portion of intestine above the occlusion and sinking of the weighted loop into the true pelvis (Merly, l. c.), or even find its way through a small opening at the strangulated spot, pass out, and leave things as they were before (Löwenhardt[6]).

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[1] Hufeland's Journal, 1836, St. 8.

[2] Ibid. 1836, St. 2.

[3] Medic. Zeitg. 1858, S. 23.

[4] Medic. Annal. III. 4, and Schmidt's Jahrb. Bd. 23, S. 184. See also *De Haën*, T. XI. p. 187, et seq.

[5] Hufeland's Journ. 1840, St. 4. See also *Morgagni*, Ep. 34, Art. 13.

On the other hand, I shall not deny that quicksilver may be of use in fæcal obstruction of the colon by exciting peristaltic action. But are not obstructions of that kind far more suitably and certainly removed by free injections of warm water or even by mild laxatives? The reported "cures of ileus by quicksilver" often refer to nothing but coprostases. We can even now declare this almost with certainty of some of them. Such are those in which, a long time after the quicksilver was given, the bowels have suddenly opened, and the whole of the mercury has been evacuated at once, together with a large amount of fæces. This could only happen if the obstruction was low down in the colon or rectum and the quicksilver had gradually collected above it, for if the ileum had been impermeable the mercury in its further progress to the anus would have been again divided into a great number of small particles and evacuated gradually. How often has quicksilver been praised when severe fæcal obstructions have been removed by the simultaneous use of enemata; how many an injection of belladonna or tobacco has owed its famous effect, not to the ingredients contained in it, but to the softening action of the water upon the fæces. It is everywhere claimed that quicksilver is especially valuable in cases of "twisting." They do not remember that twisting of the ileum is so exceedingly rare that in making a diagnosis intra vitam we need really pay no attention to it. The frequent acute twistings of the sigmoid flexure have so rapid a course—their average duration, as we saw, is only four days—that, before the quicksilver introduced by the mouth has begun to collect above it, the termination has long been decided. Furthermore, would it be rational to operate against a twisted sigmoid flexure through the mouth when we can act far more certainly and directly by enemata and inflations of air? I therefore feel justified in rejecting, with Habershon and others, the use of metallic mercury in ileus.

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[1] Schmidt's Jahrb. Bd. X. S. 14.

[2] Med. Centralztg. 1856, S. 60.

[3] Virchow's Arch. Bd. 49, S. 185.

[4] Rat. med. contin. T. III. p. 40. Compare *Pantlen's* interesting case, Württemb. Corresp.-Blatt, 1874, No. 36.

[5] *Hilton*, *Golding Bird*, Med. Chir. Trans. 1847, Vol. XXX. 1.

[6] Preuss. Ver.-Ztg. 1845, 3.

Among the therapeutical measures recommended in cases of internal strangulation are yet to be mentioned the so-called *monster-clysters.* It is evident that not only in fæcal obstructions of the colon and rectum, but also in twisting of the sigmoid flexure, lateral kinking of the ascending colon or cæcum, ileocæcal and colon invaginations, compression of the rectum or colon by a movable tumor or viscus (the mesentery of the ileum, for example), large enemata injected with a certain degree of force may be of therapeutical value. The procedure of administering large enemata at once in all cases of impermeability of the intestine deserves to be retained, for the reason also that by its aid we often reach diagnostic conclusions regarding the seat of the obstruction. For the administration of these large enemata the clyster-pump has hitherto been almost exclusively employed. Blach and Barrie (1835) advised that it should be combined with the knee-and-elbow or supine position, with the head down and buttocks raised. Recently Hegar[1] has substituted for the injection of water by means of the clyster-syringe or pump its introduction through the anus by a funnel apparatus —glass funnel, rubber tube, and rectal sound—while the patient is placed in a suitable position, which reduces the abdominal pressure as much as possible—knee-and-elbow, knee-and-head, or lateral abdominal. With the patient in such a position it is often possible to introduce four or five quarts of water into the intestine. The pressure of the entering column of water can be increased or diminished at will by raising or lowering the funnel. Injections of this kind can certainly be employed in individual cases with results as favorable as those obtained by enemata administered by means of a clyster-pump, and they have a peculiar merit in the constancy and uniformity of the pressure of the column of water, and also because the position of the patient greatly helps the introduction of large amounts of water. Attempts to act upon an obstruction in the colon or lower part of the ileum by means of injections of water can be very well combined with the opium treatment; they should be repeated several times daily and continued until the appearance of peritonitis puts a stop to all active measures. Lukewarm water should be used, not only when it is intended to soften fæcal accumulations, but *always*. Enemata of ice-water, or with turpentine, as I saw used in England, or with carbonic acid water, as Richelot recommended, excite energetic reflex peristaltic action. They are, therefore, like purgatives, and the same is true of them that has already been said regarding the treatment of internal strangulation by purging. For the same reasons I cannot speak with favor of the "ice-treatment of ileus," recommended by Grisolle and others (large compresses of ice upon the abdomen, and enemata of ice-water every two hours).

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[1] Deutsche Klinik, 1873; Berlin. klin. Wochenschr. 1874, 6. See also *Mosler*, Berl. klin. Wochenschr. 1874, 45.

It is a question whether occlusions or invaginations in the lowest part of the ileum can be directly acted upon by means of enemata. Fabricius ab Aquapendente, Riolan, Kerckringius, Haller, Morgagni, and others, came to the conclusion, after experimenting upon the cadaver, that the ileocæcal valve always, or at least in most cases, allows air and water to pass through it from the colon. These experiments have often been repeated since, most accurately by A. Hall.[1] In our time it had been entirely forgotten that many such experiments had been made formerly. A few years ago they were repeated, and naturally the same result followed. Thereupon the error against which Rahn, in 1791 (l. c., S. 2), and Videmar (De Haën, T. VIII. C. 5), had warned us, was again committed of at once applying to the living the results of experiments upon the cadaver. But in the living the tonicity of the ileocæcal sphincter, which, like that of the sphincter ani, is lost in the cadaver, approximates the two valves and makes them sufficient, and it does this the more certainly if the ileocæcal sphincter contracts by reflex action under the irritation of the pressure of the injected water against it; the opinion of Meckel, Cruveilhier, Donders,[1] and others, that the ileocæcal valve in the living body and under normal conditions prevents the return of the contents into the ileum, is entirely sound, even if experiments upon the cadaver furnish other results. If then we wish to act directly with enemata upon incarcerations or invaginations in the lower part of the ileum, it is advisable to previously relax the ileocæcal sphincter by chloroform narcosis or by large doses of opium, so as to open the way for the injected water to reach the point of occlusion. But not only under these circumstances, but also when we wish to reduce a twist, knot, or invagination of the colon by enemata, it is advisable, for more than one reason, to aid the operation by previously bringing the patient under the influence of opium.

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[1] Monthly Journ. Jan. 1846. Compare: *De Haën*, Abhdlg. Pars II. p. 82, et seq. Tom. VIII. C. V.; also *Haguenot*, Mém. de l'acad. roy. Ann. 1713; *Trauvetter*, Deutsches Arch. f. klin. Med. IV. 476.

Among those remedies which have been praised from time to time as saviors of life in ileus is electricity. Both the constant and induced currents have been recommended—one pole to be placed in the rectum, the other upon the abdomen. It is claimed sometimes that it acts by stopping the spasm, sometimes by correcting the "motus antiperistalticus," sometimes, on the contrary, by exciting antiperistaltic action below the obstruction, and in this and many other ways to have acted beneficially, and usually to have wrought an immediate "cure." The utmost that electricity can do is to somewhat favor peristaltic action in cases of fæcal obstruction, and this end can be attained by far more certain and powerful means.

Long-continued warm baths alleviate the pain, and sometimes seem not to be without a favorable influence upon certain kinds of internal strangulation.

It is very important—especially when the occlusion is situated high up, in which case the patients often die with symptoms like those of cholera, and mainly as a result of the abstraction of water from the blood—not only to quiet the continuous vomiting by opiates, and thus render it possible for the blood to take up water again, but also to aid the attainment of the same end by giving small unirritating enemata every half-hour.

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[1] Physiologie. Bd. I. 310. Compare: *Nitsche*, De valvula coli. Leipzig, 1843.

The nourishment of the patient deserves further consideration when the symptoms of strangulation last for some time. This, together with suitable stimulants, may prolong life under certain circumstances until the completion of a delayed spontaneous liberation. Here again opium is of service, for, by stopping the vomiting, it supplies the first condition that is necessary to the taking of nourishment. Sudden death from cerebral anæmia, produced by the patient's sitting up, leaving the bed, or the like, has been repeatedly observed in ileus, and corresponding cautions should be given to the attendants of the patient.

Among the measures recommended for use against internal strangulation, more empirically than with any definite rational intention, should be mentioned the "massage" or abdominal taxis, first employed by Sagar—the kneading of the abdomen while the legs are drawn up. Sagar made use of this method in a warm bath, Hutchinson, l. c., under anæsthesia, and administered at the same time large enemata of warm water. Following the practice of the older surgeons (Covillard, Sharp, South, and others) in strangulated hernia, patients with internal strangulations were also subjected to the "shaking method," sometimes with nominally favorable results.

Occlusion of the intestine by compression, can often be relieved, especially if it has occurred acutely, by the sudden displacement of a tumor (for example, when a fibroid of the uterus has been lodged in the pelvis). Puncture of a cyst, replacement of a displaced tumor through the abdominal walls, the vagina, or the rectum—Simon's method of exploration—under anæsthesia, replacement of a retroverted or flexed uterus, artificial delivery, replacement by means of sounds or forcible injections, the removal of a compressing pessary, etc., may save the patient's life. After successful replacement, a suitable posture, the wearing of a body bandage, etc., are indicated to prevent a relapse.

When diaphragmatic hernia has been diagnosticated, the treatment, so long as no signs of strangulation appear, has important prophylactic indications to meet, and real dangers may be avoided by strict dietetic measures—abstinence from badly fermented liquors containing much carbonic acid gas, constant attention to obtaining proper stools so as to avoid abnormal straining. Death has occurred in several cases as the immediate result of an emetic. If symptoms of suffocation appear after an abundant meal, after drinking liquid containing much carbonic acid, and the like, then if, as is usual, the stomach lies within the thorax, the introduction of an œsophageal sound, with aspiration of the contents of the stomach, or, if need be, puncture with a fine trocar through an intercostal space, may remove the danger.

As for the *treatment of invagination of the intestines*, we are able, in a large proportion of the cases, to make a definite diagnosis. And furthermore, the mechanical process, especially in ileocæcal and colon invaginations, is of such a kind that it is directly accessible to the influence of mechanical means of reduction. We cannot fail to be astonished that, notwithstanding this, many most irrational measures—emetics, purgatives, metallic mercury, etc.—still find advocates. At the very beginning, if the invagination makes its appearance with violent colics and vomiting, the diagnosis can almost never be made. The treatment at this period coincides with that which we rationally employ chiefly in severe colics. But if the pain accompanying the invagination is as severe as it usually is—even if it has been preceded by a constipation, lasting for several days—the prudent physician will shrink from administering drastic purges ; he will try to make enemata suffice, and, after obtaining a movement by them, he will at once begin the opium treatment. There is, in fact, no more rational treatment for the very beginning of invagination than by opium. It quiets the intestine, and prevents the increase of the invagination and the dangers associated therewith ; it favors the reduction of the swelling of the intussusceptum and the restoration of the passage ; and by the fact that it does not allow the intussusceptum to increase, it relieves the ileocæcal tenesmus, which plays an important part in the origin of most invaginations, and puts an end to the contracted condition of the portion of the intestine which contains the intussusceptum ; it alleviates and removes the pain. In such cases occurring in adults, we certainly must not work with small doses of opium ; it must be used watchfully in powerful doses—in children, of course, only in the smallest quantities and with great care. If the diagnosis of invagination can be certainly made by the detection of a tumor, passages of bloody mucus, etc., and if it can be inferred, from the position of the tumor and other facts, that we have to deal with an ileocæcal or colon intussusception—in children it is almost always the former—the indication is to attempt its reduction as soon as possible, and these attempts are the more likely to be successful the earlier they are undertaken, before the invaginated cylinders have become adherent or gangrenous. If the intussusceptum has descended into the rectum, its *manual reduction*, or *reduction by means of the sponge sound*,[1] may be tried. One must, however, have clear ideas concerning the efficiency of these methods. Neither Simon's method nor the sound can reduce an ileocæcal or colon invagination, which has got into the rectum, further than the hand and sound can penetrate, and that, as is well known, is hardly further than into the lowest part of the sigmoid flexure. Reduction by the hand or sound will therefore be of great importance, as the first step, and the rest of the invagination can then be the more easily reduced immediately thereafter by *injections of water and air*. The latter are among the most valuable means of reduction in all cases in which the intussusceptum cannot be reached by the hand or sound through the rectum. As in all these procedures it is important not to excite general contractions of the intestine, warm water should be preferred to cold.

Insufflation of air, mentioned by Hippocrates,[2] recommended by Haller[3] in invaginations, and employed successfully by Wood and Mitchel (1835, 1838), is made by means of a well-fitting bellows, or, far more conveniently, by a pump fitted with a stopcock, and has many advantages over the injection of water. Fæcal accumulations below the invagination must be removed by enemata before the insufflation.

The efficiency of all these procedures is greatly increased if the patient is under the influence of opium, or, under certain circumstances, perhaps even anæsthetized while they are put into operation. The above-mentioned postures might also be sometimes employed with success. In all attempts at reduction it must be borne in mind that they may possibly cause the most serious injury by rupture at the neck of the invagination or by reducing a gangrenous intussusceptum. Rough, forcible, ill-timed proceedings will do more harm than good here too. If the invagination has lasted for some time, if it has become fixed and chronic, or if there are signs of gangrene and impending separation, if general peritonitis or perforation is present, no prudent physician will make any attempts at reduction.

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[1] First successfully accomplished by Nyssen in 1842 (Fricke's and Oppenheim's Zeitschr. XIX.).

[2] "Folle fabrili in ventrem indito flatus immitatur," etc. *Edit. Kühn*, De Morbis Lib. III. p. 305. *Grimm's* translation, 4. Bd. S. 816. Praxagoras also employed insufflation of air, "Vento ægros per podicem replet." *Cœlius Aurelianus*, Acut. Morb. Lib. III. Cap. 17, 244.

[3] Phys. corp. humani. Tom. VII. 95. "Flatus immissus celerrime intussusceptionem dispellet."

Let us finally consider the different *operative measures* which have been suggested in internal strangulation. The mildest of these is *puncture of the intestines with a fine exploring trocar*, the object being to remove the accumulated gas, reduce the intra-intestinal pressure, and put an end to the meteorism which often causes severe dyspnœa. A fine trocar—previously disinfected by being dipped in boiling water—is thrust successively into different parts of the abdomen, which give a tympanitic sound on percussion.

The instrument does not always penetrate the intestines; it sometimes passes, as Piorry[1] showed, between the convolutions. The escape of the gas through the fine trocar can best be demonstrated by the small flame of a match. This operation, which has long been used by veterinary surgeons for the tympanites of certain domestic animals, is, if carefully performed with a fine disinfected trocar, quite without danger, according to the almost unanimous opinion of all,[2] and brings relief, which, if often only temporary, is still very considerable to the patient. It may also be remembered, that in twisting of the sigmoid flexure the torsion is often maintained merely by the meteorism, and, that when the abdomen is opened and the intra-abdominal pressure removed, the S-shaped loop sometimes (Gay[3]) springs back at once into its normal position; consequently the direct result of puncture in such and similar cases might possibly be to overcome the strangulation.

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[1] L'Union méd. 1871, No. 109.

[2] Discussion in the Bulletin de l'Acad. de Méd. XXXVI. p. 522.

[3] Lond. Path. Soc. Vol. X. 153.

Afterwards recourse was had to *larger trocars* (Fonssagrives) which were thrust, on the contrary, into the non-resonant parts of the abdomen, in order to remove the liquid fæcal masses collected above the obstruction. Puncture was afterwards united with *aspiration of the contents*. A. Paré is said to have punctured a strangulated hernia with a trocar, in order to remove the fæces; Pingray, in the seventeenth century, employed this measure several times. Van Swieten, Sharp, and others, followed the same idea, which, however, was decidedly rejected by Littré, Boyer, Schuh, and especially by Nélaton. On the other hand, Duplouy and Dolbeau published several cases of strangulated external hernia treated successfully in this way. Cazin, of Boulogne, also cured an internal strangulation by puncture and aspiration after Dieulafoy's[1] method. The latter and Demarquai used the same method, under the same circumstances, with great relief to their patients; the same happened to Henry Cooper and Gibson.[2] The two latter left the canula in place, and large quantities of fæces were passed through it for several days; afterwards the wound was enlarged by sponge tents, so as to constitute an artificial anus. Benoit[3] recommended this operation—performed usually only upon the colon and cæcum, or in the ileocæcal region—as without danger (?).

Finally, we have to consider some of the serious operative measures which may be employed in occlusion of the intestines when the chances of a favorable spontaneous termination of the affection grow less and less. My work would extend far beyond its established limits if I should treat in detail this subject, full statistics of which lie before me. The operations to be considered are:

1. *Laparotomy*, undertaken with the view of seeking for and removing the obstruction which causes the occlusion.

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[1] Traité de l'aspiration des liquides morb. Paris, 1873, p. 188.

[2] Brit. Med. Journ. 1857, p. 147. Gaz. hebdom. 1857 and 1858.

[3] Thèse de Strasbourg, 1869. See also the debates in the Soc. de Chirurgie de Paris, 5. Nov. 1873. Gaz. hebdom. No. 46, p. 737, et seq.

Opinions concerning this operation, which in earlier times was known and employed for ileus alone, have always[1] differed extremely. The objections raised by the opponents of the operation are: Laparotomy, in itself an exceedingly dangerous operation, is performed in ileus upon a patient who has been exhausted by a previous extremely severe affection, and who is usually so little able to withstand it that the chances of recovery are rather diminished than increased by the operation, and the surgeon is exposed to the danger of the death of the patient upon the table. The period at which the patient and physician decide upon the operation is usually so late, that there is great probability of the existence of general or extensive local peritonitis, numerous adhesions, and gangrene of the strangulated portion, which render the liberation of the "volvulus" impossible. Furthermore, during the search for the obstruction the intestine may suddenly tear and the fæces pour out into the peritoneum, as happened to Tessier, Fergusson, Billroth, and others. The surgeon has often failed entirely to find the obstruction, on account of the numerous adhesions binding all the convolutions together (Stein, Pirogoff, Dupuytren, Simon, Hutchinson). Finally, pathological anatomy teaches us that the volvulus is often so extraordinarily complicated, that it can be unravelled only by a careful study of the specimen after its removal from the cadaver. Another objection is the uncertainty of the diagnosis. Cases are known (Wulff,[2] Messer[3]) in which acute peritonitis or a previously latent stricture has suddenly given rise to all the symptoms of acute incarceration. The sup-porters of laparotomy maintain, on the other hand, that the medical treatment of ileus furnishes the worst possible results; that very often the death of the patient can be almost certainly predicted if there is any further delay; that the cases of spontaneous recovery at so late a period are only exceptional; and that the operation is the last attempt, the only means that still offers some chance of saving the life. It is true that the operation is often performed too late; but this is no argument against laparotomy as such, but only a warning not to allow the favorable period for the operation to pass by. The uncertainty of the diagnosis does not prevent the operation; we have only to make the incision in the linea alba if the seat of the strangulation is unknown. If the obstruction is not found, if the volvulus is gangrenous or inextricable, the establishment of an artificial anus is always in order. If peritonitis is found, it is no cause of reproach for having undertaken the operation; rather is the other reproach avoided, that of having excited it. Buchanan, Hancock, and others even believe that the cleansing of the peritoneal cavity in such cases is an advantage to the patient. Ovariotomy is referred to, the mortality in which, at first forty, is now only twenty per cent. (Spencer Wells), and it is argued, as in this operation, so, too, would the results of laparotomy in ileus gradually improve, if it was performed earlier and more frequently. It is pointed out that we have learned that the peritoneum is less to be feared when the operation is performed in a cleanly manner, and care is taken to disinfect the instruments and hands, and provide for the escape of the "septic products of the wound" (Marion Sims). Finally, statistics have been collected, and have shown that favorable results of laparotomy have not been so very uncommon; but it has not always been remembered that it is more common to publish the cases that end favorably. Adelmann found that laparotomy in ileus gave a mortality of fifty-four per cent.; Delaporte, of forty-three per cent. My larger statistics of seventy-nine cases give fifty-five deaths—seventy per cent. The most favorable cases for laparotomy, as it is easy to understand, are those of strangulation by false ligaments and internal hernias; incarcerations by a diverticle and the vermiform appendix, or twistings, are less favorable;

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[1] C. Aurelianus relates that Praxagoras recommended opening of the abdomen in passio iliaca, "magnificam autem mortem quam curationem maluisse" (Acut. Morb. Lib. III. C. 17, 274). The debates in the Paris Soc. de Chirurgie upon this subject, at the end of the last century, are interesting. Boinet says that the members of the Society, after debating for a long time, came to the conclusion "that it was better to leave the patient affected with ileus to Providence, even if the case was hopeless, than to endanger the profession and authority of physicians by performing laparotomy." Even in the old Indian therapeutics of the Brahminic period, opening of the abdomen and intestines in desperate cases of ileus is mentioned. (*Haeser*, Geschichte der Med. I. Bd. 1. Lief. S. 23, 30.)

[2] Petersb. med. Ztg. 1867.

[3] Lond. Path. Soc. XI. 110.

and least favorable of all are actual knots, adhesions, kinks, and conglomerations. If we find an obstruction by a gall-stone, foreign body, etc., or a stenosis, the establishment of an artificial anus at the obstructed point can alone be undertaken, unless the gall-stone or foreign body is lodged near the cæcum and can be forced into the colon. Laparotomy should, on no account, be performed if the symptoms of impermeability have developed gradually, or have been long preceded by those of stenosis. If the symptoms indicate occlusion of the upper portion of the small intestine—retracted abdomen—and if the impermeability has occurred acutely, then, if an operation is determined upon, there can be no question of anything but laparotomy. It should be rejected in chronic invagination, as also in stenosis ; while in acute intussusception the chances of obtaining a cure by other means are so great, that the surgeon should seldom decide to perform laparotomy. Only in those rare cases in which acute invagination leads to an early and permanent occlusion, can laparotomy be thought of for the relief of invagination, and in fact favorable results have been obtained under such conditions.[1]

2. In case it is found that the volvulus cannot be withdrawn, or that it is gangrenous, it has been recommended to excise it completely (enterectomy), and then either to unite the two ends by sutures (Maisonneuve), or to insert the upper end through an opening into the cæcum (Hacken), and to ligate the other: *entero-anastomosis.* This operation was performed successfully by Reybard[2] in 1844, and twice with a fatal termination by Maisonneuve, and now, very properly, is entirely given up.

3. *Laparo-enterotomy.* Maunoury, of Chartres, was the first to call attention, in his well-known thesis, in the year 1819, to the establishment of an artificial anus in the ileum, in cases of internal strangulation ; but he limited it, as did Maisonneuve also at first, to those cases in which, after laparotomy had been performed, the obstacle could not be found or removed. Nélaton

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[1] *Hutchinson*, Verhdlg. d. I. Congresses d. deutschen Gesellsch. f. Chir. Berl. 1872. *Wilson*, Americ. Journ. 1836, 18. *Fuschius*, Journ. f. prakt. Heilk. 1825. See also *Adelmann*, l. c., p. 61, and my work upon Invagination of the Intestines, l. c., p. 45.

[2] Gaz. méd. de Paris, 1844, p. 499.

was the first to reject, entirely, the search after the seat and nature of the obstruction with the intention to remove it, and, without giving any attention to this, to confine his efforts to the establishment of an artificial anus in the ileum above the obstruction, and thus allow the escape of the accumulated fæces, leaving the strangulation itself to its fate. The advantages and disadvantages of this method—the most suitable name of which is *laparo-ileotomy*—are apparent. The advantages are, that the peritoneum is injured to a very slight extent, and, as the fold which first presents itself at the opening is at once used for the artificial anus, the entrance of air, or, as we now say, the entrance of elements capable of exciting inflammation, into the abdominal cavity is prevented. The operation is far less dangerous than laparotomy, and easy of execution, even by unskilled surgeons. Its disadvantages are, that the cause of the occlusion, the strangulation, remains, and that peritonitis, starting from that, gangrene, and perforation, the latter accompanied still by the escape of fæces, may ensue. On the other hand are the disadvantages of an artificial anus, which must, perhaps, be borne until the end of life—a disadvantage, which, it is true, cannot be compared with the preservation of life, and can be made very tolerable, as we know, by means of suitable apparatus. The danger that a portion of the intestine lying below the obstacle may be made use of in the artificial anus, can scarcely be said to exist, for the upper portion can not only be recognized by its distended condition, but it projects spontaneously into the external incision. The artificial anus may be made so high up in the ileum that the shortness of the portion which remains applicable to intestinal digestion and absorption is incompatible with the preservation of life. This is necessarily the case when the occlusion itself is situated high up.

If, therefore, the insignificance of the meteorism, limited, perhaps, to the meso- and epigastrium, indicates an occlusion situated high up, or if it is shown by the retraction of the abdomen that the obstacle occupies the duodenum, possibly, or the uppermost part of the jejunum, laparo-ileotomy should be rejected, and if any operation is undertaken, it should be laparotomy with a search for the obstruction. The more marked and general the meteorism, the greater the chance of success in laparo-ileotomy. Most occlusions occur in the lowest part of the ileum. One of the superior loops may unfortunately present itself in the incision, but this would be an extremely rare exception if we should follow Nélaton's advice, based upon theory and experiments upon the cadaver, and always perform laparo-ileotomy in the right iliac region with an incision from one to one and a half inches above, and parallel with, Poupart's ligament. Nélaton first performed the operation of ileotomy in 1840, and subsequently six times, thrice with success. In Germany, Thüngel was the first (1853) to perform it successfully. The commendations of Wachsmuth, Adelmann, and others have established its precedence over laparotomy. Still, I think stress must be laid upon the above limitations and conditions. In chronic invaginations, if an operation is to be performed, laparo-ileotomy is the only rational one, because it offers the chance of a subsequent separation of the intussusceptum and closure of the artificial anus.

When the obstruction is situated in the ascending, transverse, or upper part of the descending colon, instead of laparo-ileotomy, the opening of the cæcum from within the abdominal cavity, laparotyphlotomy, has been recommended, an operation which was first performed by Pillore, at Rouen in 1797, and in all hitherto six times, to relieve occlusion of the intestines. Littre advised the opening of the sigmoid flexure in the left iliac fossa from the abdominal cavity when the seat of the occlusion is in the rectum or lowest part of the sigmoid flexure, "Littre's colotomy," "laparocolotomy" (first performed by Duret in 1793). For an occlusion situated higher up in the sigmoid flexure, Fine (1797 at Geneva) made the incision midway between the eleventh rib and the crest of the ileum, and laparocolotomy in the descending colon. The same object, opening of the descending colon, can be attained by an extra-peritoneal operation in the lumbar region, Amussat's[1] or lumbar colotomy. In a few cases lumbar colotomy has been performed upon the ascending colon, "right-sided," a method which in any case is to be preferred to laparotyphlotomy. The question, so long debated, of the relative merits of Littre's and Amussat's colotomy, can now be easily answered; in adults, Amussat's colotomy, notwithstanding its greater operative difficulties, and notwithstanding the greater objections to an artificial anus in the lumbar than to one in the iliac region, is decidedly to be preferred. In children, on the contrary, Littre's plan is to be adopted, and Amussat's rejected.

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[1] Improperly known also as Callisen's method.

If now we group laparo-ileotomy, -typhlotomy, and -colotomy, under the general name of laparo-enterotomy, my statistics (excluding cases of Littre's colotomy undertaken for the relief of atresia ani) contain forty-five cases of laparo-enterotomy performed on account of occlusion of the intestine. Of these, twenty-eight ended fatally, a mortality of sixty-two per cent. (The mortality, according to Adelmann, was fifty-six per cent., Whitall sixty per cent., Delaporte thirty-four per cent.). Nélaton's laparo-ileotomy and Littre's laparocolotomy give almost the same percentage of mortality (sixty-one per cent.); laparo-typhlotomy was more unfavorable. Thirty-six cases, which I have collected of Littre's colotomy in children, give a mortality of only fifty per cent., proving that this operation yields better results upon children than upon adults. I refrain from discussing the reasons.

Lumbar colotomy by Amussat's method has been performed for several years with increasing frequency, especially by the English surgeons (Adams, Ward, Bryant, Curling, Allingham, Maunder, Savory, Laffan, and many others), and has given unexpectedly favorable results, as is shown by the latest statistics, Erskine Mason's, according to which one hundred and two cases of this operation (upon adults) gave a mortality of only thirty-three per cent.

The therapeutics of constriction of the intestines, if we exclude constrictions of the rectum that are open to surgical treatment, are mainly prophylactic and palliative: prophylactic to this extent, that it must seek to avoid everything that may possibly obstruct the stenosis and lead to impermeability. Such patients should keep their bowels as regular as possible—mild laxatives continued for a long time are suitable; and they should avoid the danger of occlusion by observing proper dietetic regulations, such as abstinence from food that produces a large amount of fæces, avoidance of indigestible substances (fruit pits, etc.), and a too exclusively vegetable diet. If to the symptoms of insufficient evacuation of the contents (which may co-exist with diarrhœa) there are now added severe colics, meteorism, great prominence and active movements of the intestines, as seen through the abdominal walls, I have seen under such circumstances the best results follow the early use of opium, because the activity of the bowels is thereby regulated and proper evacuations obtained.

In stenoses which cause the patient intense pain, as in certain stenoses of the rectum, for example, those due to compression, or even in cancer of the rectum, an artificial anus has been repeatedly established of late by lumbar colotomy, and in other cases by ileotomy after Nélaton's method, and the result, as the English records of the last decade clearly show, has often been to at least prolong life and render it more endurable.

Finally, with reference to chronic coprostasis and habitual constipation, under which title actual stenoses are also found, it is not my intention to detail the innumerable remedies and measures that have been recommended. Many dietetic regulations with reference to the choice and avoidance of certain articles of food and drink, suitable modifications of the most varied kinds in the mode of life and habits of such patients, the long-continued use of mild laxatives and various mineral waters, of fresh and salt baths, regular attendance upon mineral springs, baths, and cold-water cures, the Swedish movement cure, and electricity, and, finally, a great number of the most different drugs would have to be critically examined. The correct and successful choice of these remedies depends upon the study and recognition of the causes of the habitual constipation, upon the clearness of our conceptions of the mode of action and efficacy of the different therapeutical measures, and upon a consideration of the peculiarities of the patient himself.

INTESTINAL PARASITES.

HELLER.

INTESTINAL PARASITES.

Bamberger, Krankheiten des chylopoëtischen Systems. II. Aufl. Erlangen. 1864, p. 431.—*van Beneden*, Mém. sur les vers intestinaux. Paris. 1858.—*Bremser*, Lebende Würmer im lebenden Menschen. Wien. 1819.—*Cobbold*, Entozoa. London, 1864, and Supplement, 1869.—*Davaine*, Traité des Entozoaires, etc. Paris. 1860.—*Küchenmeister*, Die in und an dem Körper des lebenden Menschen vorkommenden Parasiten. Leipzig. 1855.—*Leuckart*, Die menschlichen Parasiten. Leipzig u. Heidelberg. 1863.

Reference will be made in the following pages, at the proper places, to the more important works on this subject, from amid the mass of ancient and modern literature. For further information as to the older literature, consult *Davaine*.

Introduction.

The history of intestinal parasites reaches back to the very earliest times ; there were then, however, but few forms known, and the greatest ignorance prevailed with regard to everything concerning them. The Greeks distinguished three sorts only, the tape-worm (*ἕλμινς πλατεῖα, ταινία*), the round-worm (*ἕλμινς στρογγύλη*), and the thread-worm (*ἀσκαρίς*), which three species we find mentioned in Hippocrates and Aristotle. Some of the Romans, on the other hand, as for instance Celsus and Pliny, recognized only two species, broad- and round-worms ; while Galen again distinguishes three forms. Finally, the Arabian physicians looked upon the single specimens of the tape-worm as a separate species, and this last idea was pretty generally held up to the seventeenth century. It was not till more modern times, nor indeed till the present day, that more accurate information was acquired, both as regards the immense variety of forms and as to their natural history. It would lead us too far to consider at present the many speculations that have been made about their nature and origin, though such an investigation could not fail to be most instructive.

Of the animal parasites which take up their abode in man, and whose number is about fifty, twenty-one inhabit the intestinal canal. It is probable, however, that our knowledge in this respect is not yet complete, but rather that the number will be increased, especially by observations made in countries outside Europe, as soon as those making them are possessed of the necessary preliminary information.

Thus Bilharz has readily found out several new parasites in Egypt. Even at home such discoveries may from time to time be made, and there is now in the Pathological Institution in Erlangen a specimen of a still undetermined species of Tænia which was expelled from a child.

Of the twenty-one intestinal parasites[1] three are infusoria, nine belong to the tape-worm class, two to the suctorial, and seven to the round-worm class.

I. INFUSORIA.

1. Cercomonas intestinalis.
2. Balantidium coli.
3. Psorospermia.

II. TAPE-WORMS.

4. *Tænia solium.*
5. " *saginata* (or mediocanellata).
6. " cucumerina.
7. " nana.
8. " flavopunctata.
9. " Madagascariensis.
10. *Bothriocephalus latus.*
11. " cordatus.
12. Undetermined species (in Erlangen).

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[1] The work of *Lösch*, on "Amœbæ in the Intestines" (Virch. Arch. 65, S. 196), which tends to corroborate a doubtful observation of Lambl's, arrived too late for me to make use of it.

III. LEECH TRIBE.

(Trematodæ.)

13. Distomum crassum.
14. " heterophyes.

IV. ROUND-WORMS.

15. *Ascaris lumbricoides.*
16. Ascaris mystax.
17. *Oxyuris vermicularis.*
18. *Trichocephalus dispar.*
19. *Trichina spiralis.*
20. *Anchylostomum duodenale.*
21. Echinorrhynchus gigas.

Of these twenty-one parasites only the eight whose names are printed in *larger type* are peculiar to man. The rest are either parasites of the domestic animals found occasionally in man, or have been found so seldom in the human intestine (in some cases only once), that some other animal must be looked upon as their true host.

Cobbold's Tænia lophosoma, as well as Küchenmeister's Hottentot Tænia, are only deformed examples of the ordinary Tænia, and will be noticed under that head.

Küchenmeister has suspected the presence of Tænia echinococcus in the small intestine of man, but it has never as yet been demonstrated there (vide Vol. III. p. 562 of this Cyclopædia).

Our knowledge of the *geographical distribution* of the intestinal parasites is, in spite of numerous observations scattered here and there through our literature, very imperfect. For up to the present time sufficient attention has nowhere been directed to the subject; still less are we possessed of any exact statistics of the relative frequency of the several species in different countries.

Quite as general, and therefore as little deserving confidence, are the statements as to the frequency with which each form is met at different *ages* and in different *sexes*. My experience, founded, it is true, on a comparatively small number of cases, tends flatly to contradict some of the views held up to the present time, especially about round-worms. Thus the general idea is, that round-worms and thread-worms are, out of all proportion, more common in children ; the following table of statistics shows that they are quite often, indeed oftener, met with in adults.

The following table of statistics, founded on post-mortems made by Professor Zenker, in Erlangen and Dresden, is taken from K. Müller's Dissertat. inaugur. Erlangen. 1874.

I. Erlangen. 1862–1872.

	Number of Post-mortems.	*Cases of Ascaris.*	*Oxyuris.*	*Trichocephalus.*
Men	845	93 = 11%	113 = 13.4%	107 = 12.7%
Women	513	81 = 15.7%	57 = 11.1%	69 = 13.5%
Children (under 15 years)	397	53 = 13.3%	43 = 10.8%	19 = 4.8%
Total,	1755	227 = 12.9%	213 = 12.13%	195 = 11.11%

In this table the results of 138 post-mortems made on patients at the Insane Asylum, which gave much higher percentages, are not included. In every one of these cases one or more varieties of worms were found ; in 135 cases round-worms were found in the following combinations:

1. *Ascaris lumbricoides :* alone 14
With Oxyuris 6
" Trichocephalus 8
" Oxyuris and Trichocephalus 11
39

2. *Oxyuris vermicularis :* alone 35
With Trichocephalus 26
Total, 61

3. *Trichocephalus dispar :* alone, 35.

Ascaris 39, Oxyuris 52, Trichocephalus, 80.

II. Dresden, 1852–1862.

	No. of Post-mortems.	*Ascaris.*	*Oxyuris.*	*Trichocephalus.*
Men	1164	95 = 8.1%	24 = 2.1%	35 = 3%
Women	739	70 = 9.5%	19 = 2.5%	11 = 1.5%
Children	36	15 = 41.6%	0 = .0%	4 = 1.1%
Total,	1939	180 = 9.1%	43 = 2.1%	50 = 2.5%

Since I have entered upon my appointment at Kiel, 782 post-mortems have been made at the Pathological Institute. As a general rule attention was paid to the absence or presence of worms, and when they were found it was noted down. The following statistical survey, though founded on a comparatively small number of cases, is of importance on account of the present great dearth of reliable material.

Number of post-mortems from October 1, 1872, to September 30, 1875	782
Untrustworthy, . . . 52 Under six months old, 119	171
Leaving for our purpose	611

Of these	266	were men	and 126 or 47.3	were infested with parasites.
	194	women	96 or 49.4	
	151	children under 15	69 or 45.7	
Total,	611		291 = 47.6%	

Ascaris lumbricoides: alone		32
	Was found with Oxyuris	16
	" Trichocephalus	26
	" Oxyuris and Trichocephalus	34
	Total,	108

2. Oxyuris vermic.: alone		57
	With Ascaris	16
	" Trichocephalus	35
	" Ascaris and Trichocephalus	34
	Total,	142

3. Trichocephalus dispar: alone		90
	With Ascaris	26
	" Oxyuris	35
	" Ascaris and Oxyuris	34
	Total,	185

Thus, in the 611 post-mortems:

Ascaris lumb.	was found in	17.7%
Oxyuris vermic.	"	23.2%
Trichocephalus dispar.	"	30.6%
Round-worms	"	47.6%

Age of the youngest individual, with: Ascaris, 2 years; Oxyuris, 5 weeks; Trichocephalus, 11 months. The age of the eldest with: Ascaris, 78 years; Oxyuris, 82 years; Trichocephalus, 89 years.

Now, though great attention was given when searching for the worms, nevertheless the above figures are below the truth; for some of the post-mortems were made by inexperienced hands and eyes; further, in private houses the intestines were, for other reasons, not all opened, or the surroundings, especially with regard to light, were often most unfavorable ; and finally, single examples, especially of Oxyuris, often escaped observation.

Statistics, founded on post-mortems, are of less use for determining the frequency of tape-worms ; for a person afflicted with one of these parasites soon becomes aware of it and takes steps to rid himself of his guest.

In Thüringen, according to Conta[1], one inhabitant out of every 3,315 has a tape-worm ; while in the four medical districts of Eisenach, Apolda, Jena, and Weimar, one out of every 486 is similarly afflicted.

A calculation, based on the amount of the most common medicines used for tape-worm, which were sold by the apothecaries in the town of Hannover[2], during a period of three years, gives an average of 1,841 tape-worm patients annually. If we subtract two-sevenths of this number as occurring in country people, there remain 1,431 tape-worm patients in a town of about 70,000 inhabitants, or one in every forty-eight, which is something over two per cent.

Accounts that now and then find their way into our literature seem to indicate that tape-worm is quite as frequent in other places as it is in Kiel and Erlangen. Thus Lambl,[3] when making extensive observations on fæces in Prague, very frequently met with the egg of the three varieties of round-worms. Kyber,[4] of Dorpat, says: "The frequent presence of round-worms in the intestines of those who had died of cholera, did not escape the older investigators. The number that is sometimes found is quite astonishing." The rare and peculiar form of the fruit of the cholera fungus, said by Hallier "only to be found in the intestine," he holds to be nothing but the eggs of the Bothriocephalus. Among 482 post-mortems made by Cruse,[5] at Dorpat, Ascaris lumbricoides was found in 9.9 per cent., of whom 8 per cent. were men, and 12.4 per cent. women; Bothriocephalus latus he found in 6 per cent. Ascaris lumbricoides must be very common in Aix-la-Chapelle, or Debey[6] would certainly not have found their eggs so constantly in the evacuations of cholera patients, as to be led to think they were a cholera fungus. Similarly the plates that have been given by Swayne, Budd, and Brittan,[1] of a cholera fungus which they found in the evacuations of cholera patients, show that, for the most part, it is nothing but the eggs of the Ascaris, Oxyuris, and Trichocephalus, and prove the great frequency of these parasites in England. Out of thirteen post-mortems made by Röderer and Wagler,[2] in Göttingen, in twelve Ascaris lumb., and in six Trichocephalus were found.

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[1] Zeitschrift für Epidemiologie, I.

[2] Ueber Schlachthäuser mit besonderer Rücksicht auf die Verhältnisse in Hannover. 1865, p. 32.

[3] Prager Vierteljahrschrift. 61. p. 43, 1859.

[4] Dorpater med. Zeitschrift, III. p. 75. 1873.

[5] Dorpater med. Zeitschrift, II. p. 315.

[6] Deutsche Klinik. 1867. Nos. 1 and 2 (and 5).

It is much to be wished that wherever the opportunity is afforded of observing whether parasites are present or not, the slight trouble necessary may not be shirked. A glance into the cæcum will tell us at once whether Oxyuris or Trichocephalus is present; but it is necessary to open the whole of the small intestine to find round-worms or tape-worms.

As to the mutual effect that the different species have on each other, it is certainly not true, as was formerly held, that some species mutually exclude each other. On the contrary, we frequently find several different species in the same intestine; this is especially true of the three common round-worms, the Ascaris, Oxyuris, and Trichocephalus, which are very often found in company (vide p. 672, and 673). In like manner we may meet tape-worms in company with these, and even the different varieties of tape-worms may take up their abode, side by side, in the same intestine.

I have often observed in patients under treatment for tape-worm, that numbers of Ascarides were expelled along with the segments of the tape-worm; less frequently I have observed the expulsion of round-worms; but never, up to the present time, Trichocephali; this last is obviously due to the greater power of resistance possessed by the latter.

In animals we often see numberless round-worms and tape-worms together.

Etiology.

The first condition necessary for the development of a parasite in the intestine is of course the introduction of eggs, embryos, or, in the case of parasites which change their abode, of young forms; or, where there is an alternation of generation, of the animal in the corresponding (earlier) stage of its development. The means by which they find their way into the digestive canal are very various. In the great majority of cases they are introduced with the food, which may have been contaminated with them, or which may be the natural habitation of the young forms or of the animal when in a previous stage of development.

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[1] London J. of the Med. Sciences, 1849. I. p. 1048, pl. 1 and 2. (*Robin*, Végét. parasit. p. 676, pl. 12—but especially Lewis. A report of the microscopic objects found in cholera evacuations. Calcutta, 1870.)

[2] De morbo mucoso. Göttingen. 1762.

The question of spontaneous generation from the unhealthy humors, or from metamorphosis of portions of the intestine or its contents, naturally does not nowadays demand our consideration. Similarly we speak no more of helminthiasis; there is no longer such a thing as a tendency to breed worms, a worm diathesis, a morbus verminosus, as the older writers used to call it, who even went so far as to talk of a worm disease without worms being present. Of course we must not leave out of consideration the many personal and local conditions which are either favorable for the acquisition of a parasite, or are, on the contrary, hostile to it. All such conditions are, however, to be looked on as external, and not caused by any diseased condition of the body. Many unhealthy conditions of the digestive canal may have an influence, but it is more a negative one. Thus, in cases where there is diarrhœa, the germs of the parasite seem to find it harder to obtain the conditions favorable to their further development, perhaps from not getting the repose necessary to rest and fix themselves.

Of 116 persons, who, in the year 1874, were found to be free from parasites, 51 suffered from some intestinal complaint, which, in a large majority of the cases, was tubercular ulceration.

The idea of an *hereditary tendency*, as was formerly seriously held, can now no longer be entertained. The accounts that have been given of finding intestinal parasites in newly-born babies, are founded on an error, for either the things supposed to be parasites were not such, or they did not come from the newly-born infant.

The *occupation* of the individual has considerable influence in predisposing to the acquisition of certain species of worms. Thus persons who are engaged in the preparation, sale, or dressing of animal food, are especially afflicted with tape-worm. The degree of culture that people possess, and the habits and customs dependent on it, have a very powerful influence. For cleanliness in general, and especially with regard to eating and drinking, is directly dependent on it. And thus we see that nations which possess but little cultivation suffer greatly from parasites of every description ; so much so, indeed, that among them it is quite the exception to find any one who is quite free from them.

The *seasons of the year* seem to influence, in many ways, the acquisition of intestinal parasites ; the changes in the habits of life, dependent on the variation in the weather, which have most influence in temperate and cold climates, are of less moment, but must not be looked on as of no importance. Of much more importance is the influence heat has in the development of the eggs of many species of worms. Thus it is only after being exposed for a considerable time to the influence of warmth that the eggs of the round-worm show any progress in development, while they remain quite stationary if placed in a low temperature.

General Pathology.

Intestinal parasites played a most important part in the pathological anatomy of the ancients, owing to the great ignorance of that branch which then prevailed among the profession. For, on the one hand, while they were unable to make out any other change in the normal condition of the parts, the intestinal parasites were, on account of their number and frequency, found in nearly every case. What could therefore be more natural, than, in the absence of any other ascertainable cause, to hold them responsible for all the symptoms of the disease ; and thus diseases of every description were put down as worm diseases. Such ideas nowadays pervade only the country people, though here and there they still find sympathizers amongst the profession. Now, indeed, we are in danger of running to the other extreme, with Bremser and others, who consider the parasites as perfectly harmless guests, though there is little likelihood of our going so far as to look on them as the guardian angels of children, ever ready to help them in their time of need. This idea was held by Jördens[1] and others of the older writers, in order to satisfy their own teleological wants.

In a large number of cases, the presence of intestinal parasites does not give rise to sufficient disturbance to lead to their presence being detected. Innumerable persons entertain greater or less numbers of such guests for years, without ever being made aware of their presence except by accident.

We are not, however, on this account, justified in denying, *a priori*, that they can have any bad effect. There are numerous ways in which such an influence may make itself felt. The latest investigations about the disease caused by the Anchylostomum, show us how seriously they may sometimes interfere in the animal economy.

The general drain on the system, caused by the amount of fluids consumed by the parasites as nourishment, is of less importance. The average weight of six examples of Tænia solium was ten grammes, the maximum being 19.9, and the minimum 5.1 grammes. The average weight of eight specimens of Tænia saginata was 42 grammes, the maximum being 64 and the minimum 31 grammes. Now, since a tape-worm requires at least eight weeks to arrive at maturity, the total loss to its host could not possibly exceed 64 grammes, and is therefore quite insignificant. In all probability the same is true of the other varieties, with the exception of the Anchylostomum duodenale.

The weights of Tænia, just given, are undoubtedly too small, for the worms were taken from two patients, of whom one had two and the other four. It appears to me that in such cases the worms are not so fully developed as when they occur singly. At the time, I had, however, no other perfect and fresh specimens at my disposal.

Mechanically, as foreign bodies, endowed, in addition with the power of voluntary motion, the intestinal parasites must produce a certain amount of irritation of the intestinal mucous membrane. Nervous affections have especially been put down to their action, and looked upon as reflex phenomena. Indeed, there seems to be no reason why such phenomena should not be produced by parasites, in the same way as we see them produced both physiologically and pathologically. They are also said to cause neuralgia, which Kratz[1] has specially observed during the first few days after infection with Trichinæ. The larger species have been further accused of giving rise to intestinal obstruction, being able, it is said, when entangled into a ball, to close mechanically the whole calibre of the intestine. Davaine very properly considers this an erroneous idea ; for, cases have been known where the intestine was literally crammed with hundreds of round-worms, and still the circulation of the chyme through the interspaces was not in the least interfered with. Just as little can we admit that they have the power, with the exception of the Anchylostomum and the Echinorrhynchus, of injuring or piercing the healthy intestinal walls. Even where this is diseased, more especially ulcerated, it still remains more than doubtful if they possess this power ; and the fact, that they have been found in the peritoneum, in cases of intestinal perforation, speaks more for the frequency with which they are present in the intestine, than for their power of doing injury. In cases where perforation of the intestine has already taken place, they may prove injurious by escaping with the contents of the intestine into the peritoneum, and by their active movements preventing the occurrence of partial adhesive peritonitis, thus increasing the liability to an attack of general peritonitis.

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[1] Entomologie u. Helminthologie des menschlichen Körpers. Hof. 1802.

It is very questionable if any of the intestinal parasites are capable of doing any injury by means of chemical action, as Huber[2] supposes to be the case with Ascaris lumbricoides, and Friedreich[3] with Trichinæ.

Many other of their effects will be enumerated when treating of the different species.

It is only in the case of Anchylostomum duodenale that we can be certain of the *diagnosis* that some parasite is present from the symptoms alone. In the case of all the other varieties the symptoms are not characteristic enough to enable us to make a diagnosis, and it is only by observing the escape of the animals themselves, or their eggs, that we can arrive at certainty.

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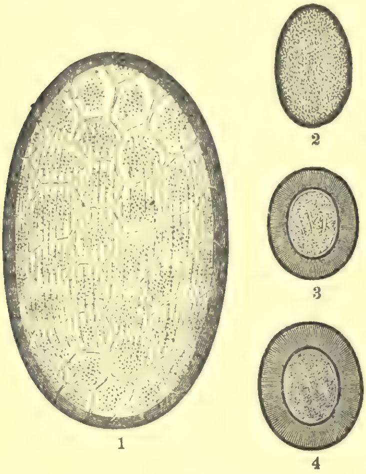
[1] Die Trichinenepidemie in Hedersleben. Leipzig, 1866, p. 82. Also in Vol. III., p. 634, of this Cyclopædia.

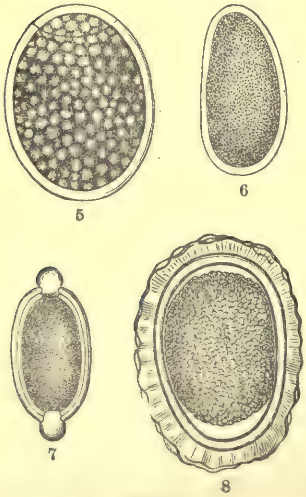
[2] Deutsches Archiv f. klin. Medicin. VII., p. 450. 1870.

[3] Ibid. IX. p. 465. 1872.



EGGS OF
1. Distomum hepaticum.
2. " lanceolatum.
3. Tænia solium.
4. " saginata.

Owing to the amazing fertility of these parasites, we have in the eggs, which they throw off within the intestine, and which are passed with the fæces, an excellent means of identification, and by which, with the exercise of a little care, we are able to diagnose with certainty their presence in a patient during life. The higher up in the intestinal canal the worm has taken up his abode, the harder it is to demonstrate by means of the microscope the presence of eggs, and it becomes easier in proportion as the worm is nearer the lower end of the intestine. For in the former case the eggs are mixed thoroughly with the fæces, while in the latter they are confined to the superficial layers, or even to the layer of mucus with which they are covered. The eggs of the different species are readily distinguished by their size and shape. The ones that demand chief attention are those of the three varieties of round-worms and tapeworms; next, those belonging to the two varieties of Distoma, which inhabit the gall ducts, and whose eggs are introduced with the gall into the intestine; and lastly, the eggs of the Anchylostomum duodenale. In the accompanying woodcuts these eggs are given, magnified about 350 diameters.



EGGS OF
5. Bothriocephalus latus.
6. Oxyuris vermicularis.
7. Trichocephalus dispar.
8. Ascaris lumbricoides.

The *prognosis* is almost absolutely favorable, for it is always possible to expel the intruder. Indeed, it would appear as if the parasite at last disappeared spontaneously without any interference on the part of the doctor, though we are as yet quite in the dark as to the age to which they attain, or what length of time they spend with their host. It is a very common thing to see round-worms especially, expelled during the course of an acute disease, and from this fact the conclusion has been drawn that the worms themselves feel the influence of the disease. It is much more probable, if the observation itself is true, that their expulsion is due to the fact, that during acute diseases the amount of nourishment taken by the patient is reduced to a minimum.

The Treatment

must be adapted to the different species, and should be founded on an intimate acquaintance with the habits and the development of each variety. Measures that will cause the expulsion of one species can be borne without inconvenience by another (see the section relating to Oxyuris).

A rational *prophylaxis* must be founded, like the treatment, on an accurate knowledge of the natural history of the parasites. Unfortunately, we have not as yet a thoroughly clear insight into the etiology and development of all the different species. Speaking in general terms, we may say that cleanliness in food and drink, as well as in the house and kitchen, is what we must rely on most to protect us from invasion. A more general diffusion of a knowledge of natural history, as well as instruction in rational diet and hygiene, is at present urgently required.

Numbers of the smaller animals have been, and still are, looked on as occasional parasites of man. It is hardly necessary to say that all such accounts are founded on intentional or unintentional deceptions, hysterical patients being especially prone to endeavor to deceive the doctor in this respect. There are, besides such so-called pseudo-parasites,[1] many other sources of error. Thus, pieces of undigested arteries or nerves of meat taken as food, the sheath in which the fruit of the orange is contained, and other indigestible substances, as well as tough strings of mucus, in cases where there is chronic catarrh of the large intestine, may all be mistaken for worms. It is not, however, very difficult to avoid such mistakes by examining the substances carefully.

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[1] *Jördens* gives a full enumeration of such pseudo-parasites in his work "Entomologie und Helminthologie, etc.," 1802.

Infusoria.

Cercomonas Intestinalis.

The Cercomonas intestinalis is a pear-shaped animal, from 0.008 to 0.01 millimeter long, and furnished with a thread-like appendage like a whip at the front of its body, which measures from 0.003 to 0.004 millimeter. From its hinder part projects a tail-like elongation, about as long as the rest of its body. In the front part of the body there appears to be a mouth. The animal progresses in irregular circles by means of a vibrating movement of its whip-like appendage. Davaine[1] distinguishes two varieties, one large and the other small. The former he observed occasionally in considerable numbers in the fresh stools of cholera patients, the latter he found in large quantities in the motions of a typhoid patient. Both varieties disappear as soon as the stools get cold. Lambl[2] appears to have observed the same animal in myriads in the jelly-like mucous stools of children. Ekekrantz[3] undoubtedly saw the same animal in two cases of chronic diarrhœa. In the first of these there were fluid stools three or four times a day, and solid motions in the intervals; in the second case the motions were slimy and were passed five or six times a day. Tham[4] also gives two cases, in which, however, the animal does not quite correspond to that described by Ekekrantz; the first case was a man who had suffered for from twelve to fifteen years from indigestion, and had constantly slight diarrhœa; during this period he had been twice attacked with cholera; the second case was a woman, who had been greatly reduced by constant purging for twelve years.

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[1] l. c. Synopsis, VI., and pp. 64, 67.

[2] Prager Vierteljahrschrift. 61, p. 43. 1859.

[3] Nordiskt medicinskt Arkiv. I. No. 20, 1869, with plate.

[4] Upsala läkareförenings förhandlingar. V. p. 691. 1870.

It cannot be determined with any certainty whether there was any connection in these cases between the disease and the presence of the animals. Probably the chronic diarrhœa produced conditions favorable to their profuse development. It is quite possible, as Ekekrantz thinks, that by their presence the disease of the mucous membrane was kept up, and periodically increased; an increase in the number of animals was always accompanied by an increase of the diarrhœa.

The presence of these parasites in any given case of chronic diarrhœa can only be determined by means of the microscope. In order to remove the animals we should, in addition to the ordinary treatment for chronic intestinal catarrh, try the effect of Hegar's method of washing out the large intestine (which will be more fully described when treating of Oxyuris), either with or without the addition of such medicines as quinine and tannic acid.

Balantidium Coli

is an egg-shaped animal about 0.1 millimetre long, with a flat abdomen and strongly arched back. It is set all round with cilia, which are particularly long around the mouth, especially on the side that corresponds to the back of the animal. At the anterior end of the body, which tapers somewhat to a point, is the opening for the mouth, which looks somewhat towards the abdominal side of the animal, and is continuous with the œsophagus, which penetrates the body of the animal to a considerable depth. At the posterior and thicker end lies the anus, which is directed, like the mouth, towards the abdomen of the animal. In the interior of the animal is the so-called nucleus, which consists of an oblong, round, finely granular body, and two elastic vesicles, which are capable of considerable alteration in their size. Alongside of these there are generally particles of the food that has been taken up. The animal possesses, according to Wiesing,[1] a very considerable power of contraction, so that it can elongate itself to twice or three times its ordinary length. It exists principally on fat globules, but he has frequently found large numbers of both red and white blood-corpuscles within it, and more rarely starch granules. He corroborates the observation previously made by Stein, that the animal propagates its species by fission, but cannot corroborate the statement of Ekekrantz[1] about the occurrence of budding. He has repeatedly seen the sexual act take place between rather small individuals, and gives a representation of it.

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[1] Nordiskt medicinskt Arkiv. III. No. 3, 1871, with plate.

Balantidium coli was first discovered by Malmsten[2] in the large intestine of man, and afterwards by Leuckart[3] in the intestine of the pig. It was afterwards twice seen in Malmsten's Clinique in Stockholm.[4] Besides these four cases, which occurred in Sweden, it has only been observed in man five times in Norway[5] and twice in Dorpat.[6]

Of these eleven cases, two occurred in women, and eight in men (the sex is not given in one case), most of whom were between thirty and fifty years of age. The occupation seems to have had no influence.

In nearly all of the cases there was chronic disease of the large intestine. Tedious diarrhœa, in some cases lasting for months and years, had greatly reduced and weakened the patients. There were exacerbations of the diarrhœa from time to time, and the stools were occasionally mixed with blood. In very few cases was the treatment followed by much improvement; in the large majority it seemed to have but little effect. In four cases the post-mortem disclosed chronic catarrhal inflammation of the large intestine, with more or less extensive ulceration, partly of a diphtheritic character. The animals were found in greater or less numbers in the evacuations, but were most numerous in the mucus that was scraped off from the interior of the intestine.

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[1] Nordisk med. Ark. I. No. 20. 1869.

[2] Hygiea. 1857, p. 491; and Virchow's Archiv. 12. S. 301. Pl. 10. 1857.

[3] D. menschlichen Parasiten. I. p. 147, 1863.

[4] *Ekekrantz*, l. c.; *Wiesing*, l. c.

[5] *Petersson*, Upsala läkareförenings förhandlingar, VIII. p. 251. 1873 (three cases). *Winbladh*, Upsala läkereför. etc. V. p. 619. 1870. *Belfrage*, ibidem. V. p. 180.

[6] *Stieda*, Virchow's Archiv. p. 285. 1866.

It is as yet not quite clear whether the presence of the animal and the disease stand to each other in the relation of cause and effect. It may merely be, that in such cases the animals found conditions most favorable for their development. Ekekrantz observed that the number of the animals was increased when the diarrhœa become more frequent, and, therefore, throws out the suggestion, that the animals by their presence might cause irritation of the mucous membrane, and thus tend to keep up the disease and cause periodical exacerbations of it.

In such cases, the diagnosis from obstinate, long-continued diarrhœa can of course only be made by the aid of the microscope.

Judging from the cases that have been observed up to the present time, the prognosis is unfavorable.

The treatment is the same as that for chronic catarrh of the large intestine; and we should, here especially, try the effect of Hegar-Simon's method of washing out the intestine, which will be described farther on, at the same time adding various medicaments to the water.

I have unfortunately been unable to procure access to a new work of Waldenström and Henschen, entitled "Upsala läkareförenings förhandlingar," IX. 1874.

Psorospermia.

Of the various parasitic forms that have been included under this head, that species alone has been found in the human intestine which is so often met with—in immense numbers—in the intestines, and more especially in the gall bladder, of the rabbit. They resemble greatly the eggs of the round-worm, possessing an oval body with a double contour, the interior of which is either filled with a uniform, coarsely granular mass, or encloses a fluid as clear as water, in which is a round, finely granular body.

According to the investigations of Eimer[1] these Psorospermia are nothing more than Gregarinæ, which have become stationary, and from which the true Psorospermia are developed by a process of fission. The Gregarina, either free or enclosed in an epithelium cell, grows up into a naked Psorospermium ; this becomes encapsuled, and then constitutes one of the egg-like bodies. This again splits up by fission into globules, which become developed into young Gregarinæ.

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[1] Die Psorospermien. Würzburg. 1870.

They have been found by Kjellberg[1] in the human intestine, within and towards the termination of the villi.

As to their importance, from a medical point of view, we can at present offer no opinion. They might, however, readily become so, if present in large numbers, and give rise to considerable disturbance. Thus we often see in our rabbit-hutches, numbers of young animals die of Psorospermia.

Tape-Worms.

History.

The tape-worm was known in the very earliest times. It is mentioned by almost all the ancient medical writers, and gives occasion to the most various speculative inquiries. It was a matter of doubt, even up to the seventeenth century, whether it should be looked upon as an independent animal being, or only as a portion of the intestinal canal, or of its contents, that had been altered by disease. All the varieties of Tænia, as well as Bothriocephalus, were grouped together under one name. Felix Plater[2] was the first (1602) who distinguished Bothriocephalus from Tænia. But its separate individuality was first secured by the truthful and accurate description and drawing given by Bremser (1811). Even then the two separate varieties of tape-worm, the "armed" tape-worm, or Tænia solium, and the "unarmed," which closely resembles it, were grouped under a common name. Goeze, it is true, remarked that there was a very considerable difference in the appearance of the two species, but he failed to bring the distinguishing characteristics sufficiently into relief. After a time differences were pointed out by Bremser, Nicolai, and others, who especially drew attention to the absence of the circlet of hooks in many tape-worms. Küchenmeister,[1] however, was the first who succeeded in finally separating the unarmed Tænia from the Tænia solium. To it he gave the name Tænia mediocanellata, which is founded on an erroneous anatomical idea.[2] We think it better, therefore, to follow the suggestion of Leuckart,[3] and to go back to the name it had before received from Goeze—viz., Tænia saginata—stout, well-fed—which exactly describes its general characteristics.

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[1] *Virchow's* Archiv. XVIII. p. 527., 1860.

[2] Praxeos med. opus. t. II. 1602.

Without, therefore, wishing in the least to disparage the services rendered by Küchenmeister, we will, in the following pages, make use of the name Tænia saginata.

We are chiefly indebted for a right understanding of the conformation of the Tænia to the investigations of Steenstrup[4] on the "alternation of generation." The tape-worm must be looked on as a colony of animals having an alternation of generation. The so-called head is the larva-like nurse; the segments of the worm—the proglottides—are the animals with sexual organs. From the head (scolex), without any mingling of the sexes, are produced the segments by a process of budding. The segments remain joined together for a considerable time; but, after they have come to maturity, they separate from the rest of the colony. The head is provided with either two or four suckers, and very frequently with a circlet of numerous small hooks. By means of this apparatus it fastens itself on to the mucous membrane of the intestine of its host. As the segments of the chain, the sexual animals, increase their distance from the head, by the development and insertion of fresh segments, they become sexually more developed, increasing at the same time in size. They are hermaphroditic, and generate eggs, in which a six-hooked embryo becomes developed. If eggs containing these embryos find entry into the stomach of a suitable animal, their envelopes become softened or undone, and the embryos are set free. By some way or other they leave the digestive canal and make their way to different parts of the body. If they now meet with conditions favorable to their further growth, "nurses" are developed in them. Should these again happen to be introduced into the intestinal tract of another animal, they fasten themselves on, and another tape-worm colony becomes developed by budding.

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[1] Deutsche Klinik. 1852. p. 9. Parasiten. I. p. 88.

[2] Parasiten, I. p. 92.

[3] Parasiten, I. p. 747.

[4] Ueber den Generationswechsel. Kopenhagen. 1842.

The time required for the development of the tape-worm colony, viz., till the sexual organs are fully developed and segments are given off, is from eight to twelve weeks. The tape-worm inhabits the small intestine, where, folded into many coils, it lies surrounded with chyme; it can move but little from its original position. Its muscular system seems, in the total absence of anything like an alimentary canal, to serve chiefly as a means of imbibing nourishment, the alternate contraction and relaxation of the several groups of muscles having an effect similar to that produced by a force-pump.

Etiology.

The ways and means by which tape-worms are acquired may, in a general way, be deduced from the history of their development. We are still, however, in the dark as to the development of Bothriocephalus, and consequently also of the way in which it gets possession of its host.

In order that a tape-worm can be developed, it is necessary that a living embryo be introduced into the stomach. This usually happens from eating flesh that is infested, and which has not been sufficiently cooked to kill the embryos or render them incapable of further development. Occasionally they may be introduced with other articles of food which have been in contact with infected meat, and to which the embryos have adhered, or which have been cut with a knife that has been previously used for cutting infected meat. Now, while these latter methods of transmission are, at all events, very rare, the possibility of infection by the former method is constantly present. The habit, or rather the bad habit, of using meat imperfectly cooked, or quite raw, which is becoming every day more common, is extremely favorable to the propagation of tape-worms.

Tænia solium is acquired by eating imperfectly cooked pork, which contains the young embryos.

We acquire Tænia saginata, on the contrary, by the use of the flesh of the ruminants, and, amongst these, the cow in particular. Tænia saginata has often been observed in those children who, for any reason, have been ordered raw, grated beef by the doctor.

The other animals which occasionally afford a habitation for the embryos of the Tænia call for little or no consideration, as they are seldom or ever used as an article of food by man.

Küchenmeister relates that in his own house embryos were found in the water in which some sausages had been washed.

Thus, Abyssinia is the classic land of the Tænia saginata, for every one, almost without exception, is infested with Tæniæ, and there it is that the use of raw, still quivering cow's flesh is looked upon as the greatest delicacy. There could not possibly be more favorable conditions than these for the propagation of tape-worms, for the greater the number of persons who have them, the more opportunity will there be for the cattle to become infected with the eggs. The custom that prevails in southern countries and among savage nations, of at once obeying the inclination to ease themselves, no matter where they may happen to be at the time, has the same tendency. Again, the greater the prevalence of embryos among the animals, the greater is the quantity of material capable of generating tape-worms in man; and so on in a constantly widening circle (circulus vitiosus).

H. Blanc[1] gives a vivid description of a banquet in Abyssinia, to which he was invited by his chief jailor, Samuel, and at which the pièce de résistance was the Abyssinian delicacy, "brindo" (brandou). "A large basket filled with flat pieces of bread was placed before us. Beside it stood a very scantily clothed female servant, the few necessary articles of whose attire were dirty and torn. With one hand she broke the bread; in the other she held a dish of sauce, which was composed of red pepper, fresh butter, salt, and gall. A man, who was also half naked, now came running up. He bore on his greasy shoulders the quarter of an ox that had that moment been slaughtered. It is considered the polite thing for the host himself to cut off the best piece, to dip it in the sauce, and then roll it up in the bread, and by a low bow to give the guest he wishes to honor to understand that the tit-bit is intended for him. As to refuse it would be looked on as an unpardonable affront, one is compelled to keep down one's disgust by a forced smile (especially if you are both prisoner and guest, and your jailor is the host), to open one's mouth, and gratefully to receive the favor. . . . The way is to cut off a a long strip of meat, carry it with the right hand to the mouth, then fix the teeth in it and cut it off close to the lips with a sharp knife, cutting from below upwards. We were permitted, on account of our awkwardness, to cut the meat, like barbarians, into small pieces, then to dip it into the sauce and roll it between two pieces of bread, making sandwiches "à la viande crue." After the first few mouthfuls we became quite reconciled to it. Flesh while fresh and warm has a pleasant taste resembling somewhat that of good oysters, and is certainly a very nourishing and readily digestible article of food. One can with ease eat two pounds, and still rise from the table with a good appetite. According to my experience, raw meat tastes better the fresher it is and the more marked the twitching of the muscles still is. Samuel, who had tasted oysters in Egypt, quite agreed with my simile, and added: "Cold brindo is just as nasty as stale oysters."

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[1] Notes méd. recueillies durant une mission diplomatique en Abyssinie. Gaz. hebdom. 1874, p. 297.

Pathology.

General Considerations.

The three varieties of tape-worm are found at every period of life, though, as a rule, most rarely in children and most frequently at the prime of life. They have not unfrequently been found even in children at the breast, especially when the latter have been fed on raw, grated meat.[1]

The female sex is afflicted somewhat oftener than the male with tape-worm, being more often exposed, from being occupied in the kitchen, to the temptation to taste raw meat.

Of Wawruch's[2] one hundred and seventy-three tape-worm patients, fifty-six were men and one hundred and seventeen women.

Certain occupations have a powerful influence in predisposing to the acquisition of one of these worms. Persons whose business is either to lay out or prepare animal food suffer much more from tape-worms than those following other vocations. Thus butchers and male and female cooks are especially liable to be thus afflicted.

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[1] *Weisse*, quoted by *Davaine*, l. c., p. 91.

[2] Praktische Monographie der Bandwurmkrankheit. Wien, 1844. (W.'s two hundred and six cases of tænia occurred in one hundred and seventy-three persons.)

Of Wawruch's 173 patients, 39 were female cooks, and one a man cook = 23 per cent.; 13 were eatinghouse-keepers, waiters, and butchers = 7.5 per cent.; and 26 were female servants = 15 per cent. Or there were altogether 79 persons thus occupied, or 45.6 per cent. Housewives who, among the country folk from whom W.'s patients chiefly came, are also constantly busy in the kitchen, are not counted in the above numbers.

It has not as yet been determined with any certainty how long, under favorable circumstances, a tape-worm can exist in the small intestines of man. The accounts we have of tape-worms, which have dwelt with the same host for twenty or thirty years, or even longer, are sadly in want of corroboration from more recent observations. Cases, of course, are not to be included in this category in which segments or longer pieces of a tape-worm have been expelled at intervals of several years, for here it is evident that the person had become possessed of several tape-worms one after the other. Bothriocephalus latus seems to be the longest lived of all. Cases are known in which a person, who had once become infested with a Bothriocephalus while living in a country where they are prevalent, has retained it for years in a country where they were unknown. Thus Bremser[1] saw a Swiss in Vienna who had one, though he had left his native land thirteen years previously; and Mosler[2] found one in a butcher, who had probably got it fourteen years previously in Russia.

Owing to the manner of their development, the seasons of the year can have no influence on either of the two species of Tænia. In the case of Bothriocephalus latus, whose embryos spend some time free in water, they may possibly have some influence, and according to Rosen[4] this worm is especially prevalent during the months of September and October.

There are many circumstances that might deceive us and make us think the season of the year was of influence. Thus the custom that prevails in many parts of

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[1] l. c., p. 94.

[2] Virchow's Archiv. 57, p. 529, 1873.

[3] *Davaine*, l. c., p. 5.

Germany of slaughtering pigs about Christmas time might deceive us with regard to Tænia solium; and the time of the year that corresponds to the fishing season might do so with regard to Bothriocephalus.

The tape-worm usually occurs singly; not uncommonly there are several, even up to a dozen; more than this is much rarer. I myself disentangled twenty-eight worms from a mass that was passed by a bath-man in the Medical Clinique in Erlangen. Other observers have seen as many as forty in the same patient. Where so many are present at once they almost invariably belong to the Tænia solium species, though occasionally Bothriocephalus is met with in large numbers; much more rarely the Tænia saginata. This last may be explained by the fact that the embryos of the Tænia saginata as a rule occur singly (vide Tænia saginata).

It is also usual to meet with only one species in the same person. Cases have, however, been recorded where more than one species inhabited the same intestine at the same time. Thus Valenta[1] found Tænia solium and Bothriocephalus together; and I have seen in a butcher Tænia solium in company with Tænia saginata.

It is said that Bothriocephalus gives rise to more serious disturbances than either of the other two varieties of the tape-worm; but nothing of the sort was observed in the cases of the Swiss mentioned by Bremser.

Symptoms.

We have already discussed in a general way, in the introduction, those convulsive affections and phenomena which are put down to the presence of intestinal parasites. It now remains to consider what disturbances are specially due to the tape-worm.

Numberless persons are inhabited for years by tape-worms without their ever giving rise to the slightest unpleasant feeling; and we sometimes have our attention accidentally drawn to their presence in children which are thriving most satisfactorily, by the expulsion of segments of the worm.

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[1] Memorabilien. XIII. p. 181, 1868. Boéchat, Gaz. méd. 1874, p. 581.

It cannot, however, be denied that very many persons who are so afflicted get an indefinite feeling of something being wrong with them. Symptoms of more or less disturbance of the digestion and nutrition, and even abnormal phenomena in the nervous system, occur, without the patient being in the least able to localize his troubles. At last the escape of the segment of a tape-worm gives a hint as to the probable source of these troubles, and the patient is cured by the expulsion of the worm.

Various unpleasant sensations in the lower part of the abdomen, which at times resemble the pains of colic, are given as a specially frequent symptom. These sensations are most marked when the person is fasting, or after the use of particular articles of diet, while, on the other hand, they are mitigated by eating, and especially by the use of certain articles as food. A sensation of ravenous hunger followed by fainting, should nothing be taken to satisfy it, is said to be of frequent occurrence. Furthermore, distention of the abdomen, disturbances of the digestive canal, especially diarrhœa alternating with constipation, the feeling as of some foreign body moving about in the abdomen, with many other symptoms, have all been put down as due to the presence of a tape-worm.

With more or less right the following have been considered as reflex phenomena, or sympathetic disturbances dependent on the presence of the worm, viz.: itching about the anus, tickling of the nose, salivation, vomiting, various sorts of hemorrhages, perspiration, headaches, ringing in the ears, palpitation, cardialgia, irregularities of menstruation, and many others, but more especially nervous disturbances of greater or less severity, such as cramps, deafness, numbness, blindness, chorea, and so on.

Most of these symptoms are probably less to be set down to the worm than to some hysterical or hypochondriacal affection of the patient. Indeed, similar symptoms may with a little trouble be discovered in all persons who have a tendency to either of these affections.

It is, however, different when we get such nervous phenomena accompanying Tænia solium. For this worm inhabits the human body when in an embryo state, and is found in the most different organs. Its chief seat is the intermuscular cellular tissue, and, next to this, the brain with its envelopes, and the eye. In these latter places it sometimes leads to serious disturbances. It is therefore quite possible that the well-authenticated cases of serious disturbance of the nervous system, when a tænia solium was present, are most readily explained by supposing that a self-infection with the embryos had taken place, especially if at the same time hysteria and hypochondriasis could be excluded. This is not, however, the place to enter on this question, which will be found more fully discussed in Vol. III., p. 603, et seq., of this Cyclopædia.

Pathological Anatomy.

No anatomical change has ever been found in the human intestine that could be said to be caused by the tape-worm. We find occasionally in the dead body that the part of the intestine where a large coil of the worm has lain is somewhat redder than the surrounding parts. This appearance is, however, produced after death. For that part of the intestine in which the worm is contained sinks from its weight readily to the bottom, and thus the appearance of post-mortem hypostasis is produced. An escape of the worms into the peritoneum can, of course, only take place when the intestine has been perforated from other causes (vide also p. 679).

Schiefferdecker[1] has observed a peculiar anatomical change in the mucous membrane of the intestine of dogs which were infested with Tænia cucumerina. The Tænia lay in tunnel-like spaces, formed by the flattening out and adhesion of the intestinal villi.

Diagnosis.

Unless the diagnosis is certain, it is scarcely allowable to make such an energetic attack on the economy of the intestinal canal, as a tape-worm cure always must be. The diagnosis is, however, only certain when it is known that proglottides have been expelled. The diagnosis of the variety of worm is also indispensable. For otherwise, if a tape-worm which has a great power of resistance were present, we might not attack it with sufficient energy, and thus the patient would have to suffer a repetition of his torture; or, if a variety were present that was easy to expel, we might act in the most energetic manner without any sufficient reason.

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[1] Virchow's Archiv, 62, p. 475, 1875.

We must, therefore, first of all determine what are the characteristic differences. And foremost among them is the fact that the segments of the Tænia saginata are passed at all times spontaneously, which happens seldom or never in the case of Tænia solium. The segment of Tænia saginata are much stronger, thicker, and less transparent than those of Tænia solium, and contain, when expelled spontaneously, very few eggs. Should the segments at our disposal be, as they sometimes are, so macerated as to make the diagnosis impossible, we can readily, by means of a mild purgative, obtain a number of fresh ones. The segments of Tænia solium are much more delicate and more transparent, and even in the fresh state we can generally, as soon as the contraction relaxes, recognize the small number (9–12) of lateral branches going off from the uterus. We can preserve them, till this goes off, in a half per cent. solution of common salt. On the other hand, in the segment of Tænia saginata we can make out the numerous lateral branches, amounting in number from fifteen to twenty. This difference may be very well and readily seen if we spread out some of the segments on a glass plate, and allow them to dry. In the segments of Bothriocephalus may be seen a brown rosette (Fig. 34) formed by the uterus, by which they are easily known from those of the two varieties of Tænia. In addition to this, the segments of Bothriocephalus are seldom expelled singly, but usually several joined together at a time.

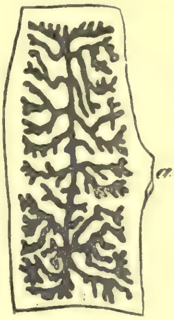

FIG. 9.

Ripe segment of Tænia solium.

a, Genital pore (magnified 6 diam.).

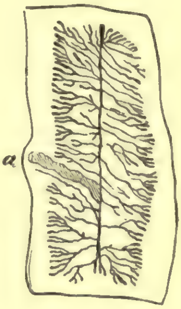

FIG. 10.

Ripe segment of Tænia saginata.

a, Genital pore (magnified 6 diam.).

It is not possible to distinguish Tænia saginata from T. solium by means of the shape of their eggs. For the eggs of both are somewhat oval, and the difference in the size is too slight and subject to too many variations.

It still requires careful observation to prove that the segments of Tænia solium are never expelled spontaneously. In the cases that are known to me, the facts were as has just been described.

The unqualified stipulation, that, before we proceed to put the patient through a tape-worm cure, the diagnosis must be made certain by the expulsion of segments of the worm, is grounded on the fact that in some cases unfortunate individuals have, for years, undergone the most violent cures without ever having had a tape-worm at all.[1]

If a patient, from whom we have received segments of a worm, has been treated for tape-worm, and no long pieces have been expelled, we are justified in doubting whether the segments we have seen were expelled from him at all, and not rather from some one else. A case has come under my own observation in which a child was greatly reduced by repeated tape-worm cures, without, however, ever having passed long pieces of a worm. Nevertheless, from time to time, segments were found in the night-chair. At last it came out that the nursery-maid had a tape-worm, and had, out of laziness, made use of the child's night-chair.

As one source of error in diagnosis must be mentioned the pseudoparasites, which not unfrequently are presented to the doctor as worms, especially by hypochondriacal men and hysterical women. If we leave out the cases of wilful deception, the things most likely to lead to error are, undigested articles of food, such as the sinews, nerves, or blood-vessels of meat used as food, and also large shreds and strings of tough mucus, in cases of chronic catarrh of the large intestine, all of which, both in form and color, have now and then a certain resemblance to worms.

Such mistakes may, however, be readily avoided by a careful examination of the objects themselves.

The *prognosis* is quite favorable in the case of Tænia saginata and Bothriocephalus latus, since we always have it in our power to expel the parasite. In the case of children and persons who are greatly reduced by disease, the prognosis is somewhat less favorable. For such persons are often unable to bear the exhausting treatment, and therefore either retain their guests or at least suffer more seriously from the after effects of the treatment.

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[1] *Davaine*, l. c., p. 62, gives the particulars of such a case.

It would also appear as if certain abnormal sensations were liable to continue, even after the total expulsion of the parasite. This is especially the case if the patient has been a long time afflicted.

The prognosis is just as favorable in the case of T. solium, as far as the worm itself is concerned, and what has been just said about the other species holds good about this. The prognosis is, however, doubtful with regard to any disturbances that may be present, and which might be caused by the system having already become infected with embryos. On this subject, see Vol. III. of this Cyclopædia.

Treatment.

There can hardly exist a disease for the cure of which so many complicated directions have been given, the most conscientious carrying out of which has been demanded as an indispensable condition for the successful result, as have been given for the cure of tape-worm. And, nevertheless, how many persons do we see suffering from tape-worm who have in vain subjected themselves to the most exhausting tortures. The enormous practice enjoyed by non-professional worm-doctors is a crying reproach to the profession. The reputation, too, that these quacks enjoy is partly founded on their success. For, on the one hand, they act without the slightest regard for the patient, and, on the other, they pay the greatest attention to the freshness and activity of their drugs. In part, however, it is founded on deception. For they persuade their patients, when only part of the worm is expelled, that they are quite cured, and know how to explain any return of the worm by the old pathological idea of a diseased condition of the intestine leading to the development of worms, or in some other way.

In carrying out the cure for tape-worm, we can readily comply with the first two conditions laid down in the old saying, "cito, tuto, jucunde," but up to the present time it has been found almost impossible to comply with the last.

The cure can only be said to be complete if the head is found, or, if several worms are present, a head for each. There is no such thing as a partial cure. The doctor should, therefore, never omit to satisfy himself of its presence in the evacuations.

The way to look for it is not difficult. First of all, pour clear water on to the evacuations, allow them to stand for ten minutes, and then pour the water off again. This process must be repeated until the water that is poured off is very little, if at all, colored. The worm, which is specifically heavier than water, almost always sinks quickly to the bottom. The head can be readily found in the last residuum, by transferring the worm, coil by coil, to a vessel of clean water, till at last all that remains is the single segments and smaller pieces. Among these the head, if present at all, will not readily escape observation. It is more rare to find the whole worm expelled in one piece.

Should we have to examine a large quantity of fluid fæces, we may first pour the greater part carefully off, and then treat the remainder as just described, for the worm and the pieces will generally be found in this portion. If we fail to find the head, we must search for it in the part that has been poured off. It is not judicious to stir the matter to be examined about with pieces of stick, for thereby the tape-worm is only broken up unnecessarily. It is quite sufficient for our purpose if we pour the water on in a good stream.

If the head be not found, either from its escaping our observation or because the patient has not conscientiously collected and kept all the evacuations, there is nothing for it but to adjourn the decision of the question, whether the cure is complete or not, for three months. If, during that time, no fresh segments have been expelled, we may conclude that the cure is perfect, as the worm only requires from eight to ten weeks to attain to full maturity.

The grounds for the success or lack of success of a tape-worm cure have not been as yet fully elucidated. We are sometimes unsuccessful though we have given exactly the same amount of the same medicine, in the same way. At other times we see a tape-worm expelled, head and all, after the administration of a gentle purgative, or sometimes even after drinking a glass of fresh water. This may in a measure depend on whether the animal's head is adherent behind a fold of mucous membrane or at its summit.

It is judicious, before commencing the actual cure, to give the patient some preparatory treatment. This has a two-fold object. First, and most important, we empty thereby the intestinal tract, so that the worm when detached may pass more quickly, and the cure, therefore, be sooner ended and the patient set free from the water-closet. Besides all this, it renders our search for the head much easier if we have fluid stools to investigate. With this object, however, only the very mildest purgatives should be given. For strong purgatives readily cause part of the worm to be torn off and expelled, and as a consequence our efforts to dislodge the part that remains may fail. This laxative treatment, joined with the administration of enemata, or, what is better, with Hegar's method of washing out the intestines (for which see under the treatment of Oxyuris), is to be carried out for two days before we proceed to the actual tape-worm cure.

During these two days the patient is to be allowed only such articles of food as do not furnish much residuum, especially meat, white bread, milk, coffee, wine, beer, tea, etc. He should at the same time carefully avoid such articles of food as pulse (Hülsenfrüchte), brown bread, vegetables, fruit containing small pips, such as straw-berries, bil-berries, currants, and also eggs: the first, because they produce a large quantity of fæces; the fruits containing pips, because they cause the worm to be broken up into small pieces, and, like eggs, make the search for the head much more difficult.

Though I am quite aware that now and then the use of fruit containing pips is alone sufficient to expel the worm, still I am not favorable to their use at the time of the actual cure.

The second object of the preliminary treatment is to administer such things as have a tendency to depress and sicken the worm, and thus render it less able to resist the actual worm medicines. Although we have no accurate knowledge on the subject, still there are a number of things that have been empirically found out to be unpleasant to the worm. Foremost among these stand garlic, salt-herrings, and onions.

On the evening of the day preceding the cure, the patient ought, therefore, to eat a plateful of herring-salad, composed of finely cut salt-herring, which has not been steeped in water, plenty of finely cut onions, and, for those that like it, garlic.

The cure itself begins in the morning with the administration of the worm medicine. It is not, however, absolutely necessary to take it fasting. We may generally permit the patient first to take a cup of coffee with a little white bread, which lessens the tendency to vomit the medicine. Of the many medicines that are all more or less effectual, the one most to be recommended is *koosso* (cusso), the blossoms of the brayera anthelmintica. An hour after the patient has taken his coffee, let him commence taking the koosso at intervals, and finish the whole quantity that is to be taken within an hour.

The quantity requisite for expelling a Tænia solium is five drachms; for Tænia saginata, which is much more difficult to expel, seven and a half drachms are necessary. The koosso may be given in the convenient form, recommended by Rosenthal,[1] of compressed balls or discs which are coated with gelatine, which hides the taste and smell of the medicine. The gelatine capsule becomes dissolved in the moisture and warmth of the stomach, and the medicine is set free, and we thus avoid the unnecessary mixture of other substances. The balls or discs are placed as far back on the tongue as possible, and, unless a person is very awkward, he will readily get them down by closing the mouth and then making the motion of swallowing. If this does not succeed, they may be washed down with a mouthful of coffee. Any inclination to vomit must be repressed, which is rendered easier by taking small mouthfuls of strong coffee or pieces of ice. Vomiting is especially to be avoided; for thereby not only does a sufficient quantity of the medicine not exert its effect, but the patient runs the risk of ripe proglottides entering the stomach, which, especially in the case of Tænia solium, may lead to an invasion of the whole system with the embryos.

Many persons find it impossible to swallow the requisite quantity of koosso in any other form than that recommended by Rosenthal, which is also admirably adapted for the administration of other medicines which are very bulky. The finely powdered mass is reduced, by means of a small special apparatus, to one-third of its original bulk, and is then covered with a gelatine capsule, in which form it may be kept unaltered for years.[2]

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[1] Berlin. klin. Wochenschrift, 1874, p. 417, and the Erlanger Sitzungsberichte for 1872.

[2] This compressed koosso may be obtained from the Schwanen-Apotheke in Erlangen.

Two hours after the patient has swallowed the last portion of the koosso, we may give him a couple of spoonfuls of castor-oil. The worm is then often expelled within an hour or two, occasionally, however, not till the evening, very rarely not till the next morning. If the worm be partially extended from the anus, we should administer an enema containing some anthelmintic.

We need have no anxiety lest the head of the worm should become fixed in the large intestine, and thus the worm be reproduced. In any case it could remain there but a short time, for the tape-worm does not find in the large intestine the conditions necessary for its sustenance, and it is quite impossible that it can mount again into the small intestine.

I have seen a large number of cases treated successfully in this way, partly under my own care and partly under that of others, especially in the Medical Clinique in Erlangen. The result in each case was proved by finding the head of the worm. In some, however, it was not found, perhaps because the patient had not kept all the evacuations, though subsequent immunity proved that the cure had succeeded. In others, the treatment failed, because some circumstance had interfered with the success, as, for example, vomiting of the medicine, too severe purging in the preparatory treatment, or the administration of a purgative immediately after the koosso. The anthelmintic must be given time to act before a purgative is administered.

The large mass of the remedy seems to have some influence on the result. For the intestine is thereby more stretched and its folds flattened out, and thus both the head and body of the worm are more exposed to the action of the medicine.

Once the cure is over, we must take compassion on the digestive tract that has suffered such rough usage, and which in some persons is excessively sensitive. The patient should therefore get some mucilaginous soup, or an emulsion with, in some cases, a few drops of laudanum, and we should not omit to draw his attention to the etiology and prophylaxis of tape-worms.

Koossin, which has been obtained by Wittstein and Bedall, of Munich, by treating koosso with alcohol, though an impure drug, is, according to a communication that I have received from my colleague, von Ziemssen, a very active preparation. For some years past in the Medical Clinique at Munich about thirty grains of "koossin" have been given in one dose for the cure of tape-worm, and it has been a very rare occurrence that the result was not all that could be desired. The preparation itself is easy to take, produces no feeling of nausea, and is on this account worthy of being more generally known.[1]

Extremely numerous are the various other drugs and methods that have been recommended, and which are worthy of a trial. Thus male fern-root, bark of pomegranate-root, kameela, benzine, saoria, oil of turpentine, picronitrate of potash, and many others have all been more or less warmly recommended.

Male fern: The active principle of this drug is probably an ethereal oil; but the evidence on this point is far from conclusive. It is usually administered in an electuary, in decoction, or in mixture (which requires to be shaken). Probably a better way would be to give it in the compressed form. The dose is from four scruples to half an ounce, according to the age and constitution of the patient. The dose is to be repeated.

The *oleo-resin* (ethereal extract) *of fern* is praised by many. It is given in doses of from seven and a half to twenty-three grains in pills, which are best made up with the root itself.

Bark of pomegranate-root: This is a very powerful medicine, but to most persons most unpleasant to take, for it causes violent pains in the abdomen, nausea, and vomiting, and a number of other troublesome indirect effects. It is given as decoction: from two to four ounces of the drug to from twenty-four to thirty-two ounces of water, divided into two to three doses; or one may macerate from two to four ounces of the drug in from one to two pints of water for twenty-four hours, then boil it down to the half, and give it to the patient in two doses, with an hour's interval between them.

Kameela (Rottlera tinctoria) has been lately very warmly recommended; from fifteen grains to half an ounce is given in two doses, generally as an electuary.

Benzine has been tried by Mosler.[2] He gives from sixty to one hundred drops in capsules, which each contain ten drops, two every hour till the worm is expelled. Its use will, however, never become general, on account of the very unpleasant general effects that it sometimes produces. It not unfrequently fails also to expel the worm.

Saoria[3] (the ripe and dried fruit of Moesa picta) has also been experimented with in Mosler's Clinique. In doses of from an ounce to an ounce and a half it is said to expel the worm completely.

Oil of turpentine[4] is without doubt a powerful remedy for worms; from one to two ounces are given in one or two doses, and the worm is usually expelled quite dead soon afterwards; it does not cause any general disturbance of the system. It is best administered in capsules, followed by full draughts of milk. Whether the freedom from unpleasant consequences after the administration of such large doses depends on the rapidity with which they pass through the intestinal canal, whereas smaller doses are often accompanied with various disturbances, has not been investigated.[1]

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[1] The best quality of koossin is to be obtained from Bedall, apothecary, in Munich. Bayer. ärztlich. Intelligenzblatt, 1867, p. 49.

[2] *Peters*, Dissert. Greifswalde, 1868. *Paulini*, Disertat. Greifswalde, 1873.

[3] *Pagé*, Diss. Greifsw. 1869.

[4] For the copious literature on this subject, see *Davaine*, l. c., p. 810.

Picronitrate of potash is recommended by Friedreich.[2] Picronitrate of potash, twenty-eight grains; jalap, nine drachms; extract of liquorice, enough to make thirty pills; one pill three times a day. Mosler[3] is not so satisfied with the results; he has specially observed general malaise for some days after its administration.

For a full account of all the other drugs and methods, I must refer the reader to Davaine, Küchenmeister, Wawruch,[4] and others.

I have myself no personal experience of the expulsion of Bothriocephalus latus. According to all accounts it is not harder to expel than the Tænia solium, and yields to the same remedies. Filix mas is especially recommended for its expulsion. In other respects the treatment is to be carried out in the way that has already been described.

It is looked on as a rule only to undertake a tape-worm cure if segments of the worm are being expelled spontaneously.

The contra-indications are: menstruation, pregnancy, great age or youth of the patient, and acute febrile diseases. Diseases of the intestinal canal are no contra-indication, as it appears such are often made worse by the presence of the worm.

We can hardly be expected, nowadays, to pay any attention to the change of the moon.

Persons who have been afflicted with tape-worm often complain of the same symptoms long after it has been quite expelled. It need hardly be said that we must not allow ourselves to be persuaded into undertaking another cure on the ground of such complaints, which, though sometimes true, are often imagined, and frequently depend on hysteria or hypochondriasis.

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[1] *Nothnagel*, Handbuch der Arzneimittellehre, p. 631, 1874. Consult also *Bartels*, Structural Diseases of the Kidney, in a later volume of this Cyclopædia.

[2] Virchow's Archiv. 25, p. 413, 1862.

[3] Virchow's Archiv. 33, p. 430, 1865.

[4] Monographie der Bandwurmkrankheiten, Wien, 1844.

The only thing to justify another cure is knowing that segments of the worm are still being passed.

Prophylaxis.

The prophylaxis presents a double problem. The first is, how to protect man from the introduction of living embryos; the second, how to guard our cattle from being invaded by them.

The first falls partly within the province of our sanitary authorities, but must also, in part, be solved by each individual.

By establishing a strict compulsory meat inspection we must prevent any infected meat being exposed for sale. Such an inspection, will, however, only be possible when we have erected public slaughter-houses.[1] Indeed, even by the most careful inspection, we can never prevent individual embryos escaping attention and being sold with the meat and eaten, for they are by no means distributed equally throughout the whole body of the animal.

Each individual must, therefore, guard himself against acquiring a tapeworm by taking care not to introduce any living embryos into his stomach. A knowledge of the appearance that infected meat presents, together with a knowledge of the most elementary hygienic rules, founded on common sense, should be more generally diffused among the masses, in order that a further supervision should be exercised in the kitchen over infected meat. But above all, no meat should be eaten that has not been sufficiently exposed to heat. It is, however, only after long roasting or boiling that large joints of meat are raised to a sufficiently high temperature in the centre to kill the embryos. We may say, as a general rule,

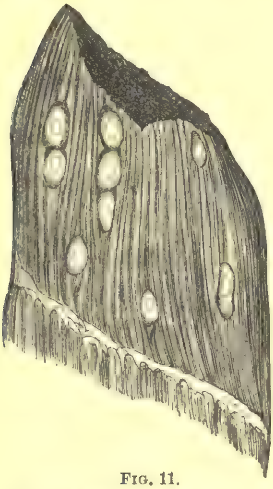

FIG. 11.

Swine's flesh containing embryos. The head can be seen through the cyst walls.

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[1] *Gerlach*, Die Fleischkost des Menschen, Berlin, 1875, could not be made use of.

that no meat should be used which still retains a red color, still less such that contains blood; and, when cut, no reddish, still less red, fluid should escape. The use of raw meat should be entirely condemned.

All this of course holds equally good for meat that has been insufficiently salted or smoked.

If we are compelled, for dietetic reasons, to prescribe raw grated meat, we must entrust the preparation of it to some person on whom we can rely, and who has been accurately instructed as to the appearance of the embryos, and, best of all, has received orders to reject every particle of meat containing a white spot, or streak, or vesicle.

The strictest cleanliness should rule, both in the kitchen and in the larder. Meat should never be allowed to lie in contact with other articles of food which are not exposed to heat before being used, nor should they be carried together in the same vessel or basket. When possible, every article of food should be washed before being used.

The second and chief part of our task is, however, to keep our animals destined for slaughter free from infection. It is quite in the power of those agriculturists who breed animals for the market entirely to eradicate the disease from their stalls.

Now, with regard especially to swine, the idea that they thrive better amid filth must be looked upon as a prejudice that is no longer entertained by skilled agriculturists. The habit that still prevails in some farm-yards, of intentionally giving the animals access to the excrement of human beings, should at once be put a stop to as thoroughly worthy of reprobation, and pigs should be so enclosed as to be unable to wallow about in the dung-heap and other filth.

Though the ruminants have no such filthy propensities, still they too should be shut out from access to human excrement. With this object, the barbarous custom of defecating in every place promiscuously should be put down with a high hand.

These precautions are not, however, by themselves, sufficient to prevent the infection of cattle. For we know that the mature segments, especially of Tænia saginata, are continually being spontaneously expelled and lost. Infection of the fodder of the animals, of the grass or of the hay, may in this way readily occur. The domestics, and especially the out-door servants, must therefore be kept under observation, and, in case any of them be found to have a tape-worm, he should be requested to have it expelled. The presence of a tape-worm is not only a constant source of danger to the health of the individual, but also to every one else in the same house with him ; and a person afflicted with Tænia solium is more to be shunned than one suffering from small-pox or the itch. It is hardly necessary to say that a tape-worm that has been expelled should not so forth be thrown aside. It should first be rendered harmless, which is best done by pouring boiling water over it.

Finally must be mentioned the danger caused by irrigating meadows with the contents of the city sewers. Cobbold was the first to draw attention to the fact that the helmintha might be disseminated in this way, by the infection of the larger animals used for slaughtering. This view met with great opposition, and indeed the careful examination of a beast that had been kept on such meadows disclosed no embryos. The danger, however, cannot be denied, for the eggs of the tape-worms would seem, from their thick, tough shells, to be likely to retain their vitality for a considerable time. An experiment that I made on a goat by feeding it on the pulp-like remains of two Tæniæ saginatæ that had been undergoing decomposition for four and six weeks during the summer, turned out successful. The limit of their power of vitality does not, therefore, seem to be as yet at all determined.

Gerlach[1] was equally successful with the eggs of Tænia solium.

Tænia Solium.

The Armed Tape-worm.

When fully developed, the Tænia solium usually measures from two to three metres, seldom more. Its head is about the size of the head of a small pin. Its shape is somewhat quadrilateral, owing to the four prominent sucking-discs. The top of the head is elongated into a conical protuberance, the rostellum, on which is placed a coronet of small hooks (vide Figs. 22, 23). The hooks are arranged in two circles, an outer, containing small hooks, and an inner, which contains larger ones. The points of all these hooks fall, however, in the same circle. The lever-like ends of the hooks by which they are attached are inserted into pocket-like depressions, which occasionally are strongly pigmented. It is very common for the hooks to fall out, especially if the worm has been expelled by medicine; but by means of a pocket-lens we can always say that they have been there. It happens sometimes also that only the hooks belonging to one circle are lost, leaving a single circlet of hooks remaining. The suckers are very movable; they are visible to the naked eye, and are protruded so far that they appear only to be connected to the head by slender pedicles.

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[1] 1. Hannover. Jahrb. 1870, p. 66.

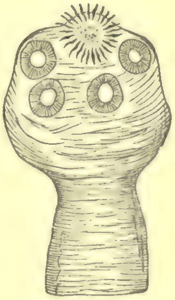

FIG. 12.

Enlarged head of the Tænia solium.

The neck, which joins on to the head, is about three centimetres long, and does not present any appearance of segmentation. The segments that are attached to it are very small and delicate, but they become larger as the distance from the neck increases. The end of each segment that is towards the head is somewhat smaller than the end that is away from it, and the small end of each segment is, as it were, let in to the larger end of the preceding one.

The mature segments measure when in a state of relaxation from nine to ten millimetres long, and from four to six mm. broad. When fresh they are usually more or less contracted, and consequently appear somewhat furrowed on the surface.

All measurements of tape-worms are likely to be inaccurate, for the size of the worm undergoes very considerable variations according to the amount of contraction present at the time. The worm is, as a rule, measured when in a state of relaxation.

The genital pore is situated on the margin and a little behind the middle of each segment, and alternates irregularly from one side to the other.[1]

The uterus (Fig. 13) forms a straight line passing down the centre of the segment, from which from seven to twelve branches are given off on each side. These branches are placed at irregular intervals from one another, and break up into branches like a tree. The segments are occasionally almost sterile, and, in consequence of the small number of eggs that are developed, the lateral branches of the uterus are much smaller and fewer in number than usual (Fig. 14).

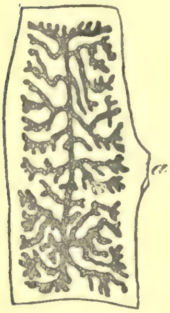

FIG. 13 (9).
Mature segment of Tænia solium. (Magnified 6 diam.)

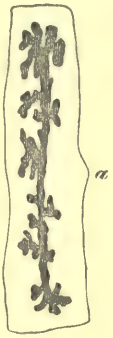

FIG. 14.
Mature, but very unfruitful segment of the same.

The other parts of the female sexual organs present a somewhat complicated arrangement. They consist of the germ-stock, which is situated in the hinder part of the segment; of the two yolk-stocks, which are situated anteriorly and to each side of the ovary; and of the "round-shaped body" (rundlichen Körper). Into this latter open the ducts of the germ-stock as well as the common duct of the two yolk stocks, into which the muscular vagina itself opens. The uterus also commences from this "round-shaped body."

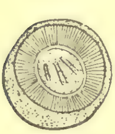

FIG. 15.
Egg of Tænia solium, containing a six-hooked embryo.

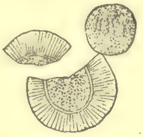

FIG. 16.
Ruptured egg of Tænia solium from which the embryo has just escaped.

The male organs of generation consist of a tolerably thick seminal-duct (Samenleiter), which is twisted like a cork screw, and which, breaking up into numerous branches, spreads itself over the anterior part of the segment. On these branches the numerous seminal vesicles sit like berries.

The eggs are roundish, and are on an average 0.032 mm.

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[2] For a fuller account of the anatomy of the Tænia, vide *Schiefferdecker*, in the Jenaische Zeitschrift, etc. VIII. p. 459, 1874.

broad, and 0.036 mm. long. They are enclosed in a very thick shell, which is composed of radiating, angular staves, arranged like a mosaic. They are often covered with a delicate albuminous envelope in which are masses of granules. The shell of the ripe egg encloses the embryo, a delicate mass of protoplasm armed with three pairs of fine, glistening hooks.

Running along the whole length of each side of the worm and reaching its whole length, is a vessel, containing a liquid, with transverse communicating branches.

Deformities occur frequently, the most common being partial or complete fusion of two consecutive segments, in which way segments are formed having several genital pores. Very often, too, the segments are stunted and full of flaws, and fissures and holes are far from uncommon. Most of these are caused simply by rupture of the immensely distended uterus, the eggs thus procuring a way of exit for themselves. However, now and then we find this splitting and perforation not confined to the mature segments, but affecting the worm throughout.

The most interesting of these deformities are the rare instances where two worms are joined together. Along the whole length of the flat surface of one worm, another worm, which is set on the edge, is joined—the second worm being usually stunted. In other cases the whole worm, instead of having two flat surfaces, has three, is in fact triangular.

An example of this deformity was found by Professor Zenker in the intestine of a person who had died of tubercles, along with thirteen properly formed worms. The head was furnished with six suckers and twenty-eight hooklets. All the worms were young examples of Tænia solium, and in none had any of the segments arrived at maturity. The deformed worm was forty-six centimetres long, and looked as if it was all wrinkled up. It had three edges, the section having the form of a Roman Y, and all the genital pores were situated on the lowest edge.

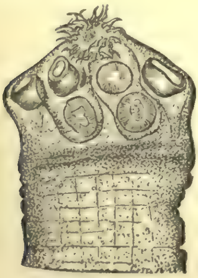


FIG. 17.

Head of a deformed Tænia solium having six sucking discs.

Many similar double monsters have been seen by Bremser,[1] Levacher,[2] Cullingworth,[3] and Cobbold,[4] and one has been described by the last-mentioned author under the name Tænia lophosoma. It would appear, however, that in each case the worm belonged to the species T. saginata. It is also plain that the worm described by Küchenmeister,[5] as found in the Hottentots, is nothing but a double monster belonging to the T. saginata species. In none of these instances was the head found. Heads with six suckers have been seen by Küchenmeister [6] in the T. cœnurus, by v. Siebold [7] in the T. echinococcus, of which he gives a drawing, and by Bremser [8] in the T. crassicollis.

The T. solium in its embryonic stage, when it is known as the Cysticercus cellulosæ, inhabits the intermuscular connective tissue and other parts of the pig. It has also been found in the

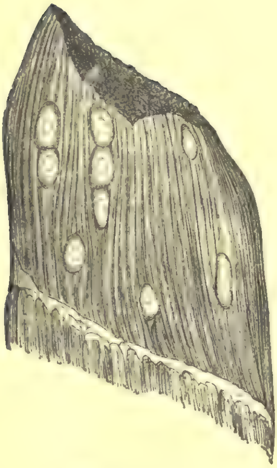
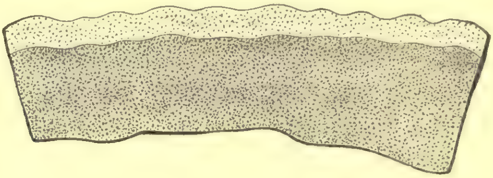


FIG. 18 (11).

Pork containing the Cysticercus of T. solium.

FIG. 19.

Flattened border of cell-wall of the Cysticercus cellulosæ.



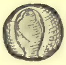


FIG. 20.

Cysticercus cellulosæ (natural size).

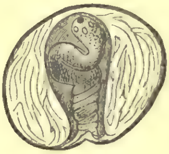


FIG. 21.

The same seen through a magnifying-glass.

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[1] l. c., p. 107, Plate III. Figs. 12–14.

[2] Comptes rend. XIII. p. 661.

[3] Med. Times and Gaz. Dec. 12, 1873.

[4] Transact. of the Pathol. Society, XVII. p. 438.

[5] l. c., p. 93. Pl. III. Fig. 14.

[6] Ibid. p. 94.

[7] Zeitschrift f. wissenschaft. Zoologie. IV. Pl. 16, Fig. 9.

[8] l. c., p. 108.

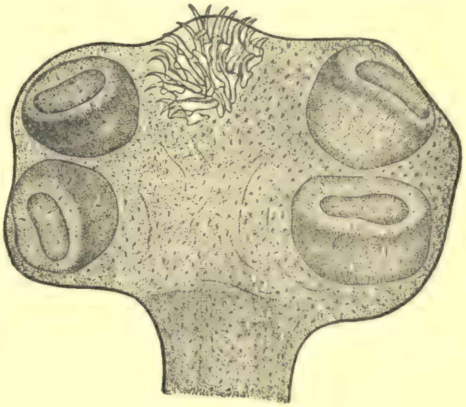
deer,[1] in the dog,[2] and in the polar bear[3] (of course when living in confinement). It is not yet certain whether it occurs in rats and monkeys or not.

For an account of its occurrence in man, refer to Vol. III., p. 601, of this Cyclopædia.

The Cysticercus cellulosæ is a thin-walled vesicle with limpid contents, seldom exceeding the size of a pea or bean. This vesicle has an irregularly wavy surface. It is usually surrounded by a capsule of connective tissue, to the inner surface of which it is closely applied. At some place on the surface of the vesicle there is a more or less well-marked depression. Inside the cyst, and visible through it, is a firm, round, whitish body, which is connected with the depression in the wall of the cyst. On opening the cyst, this body is found to be a pear-shaped sac. Inside this sac is the head of the Cysticercus, turned inside out like the finger of a glove. The head resembles, in every way, the head of the Tænia solium.

FIG. 22.

Strongly magnified head of Cysticercus cellulosæ.



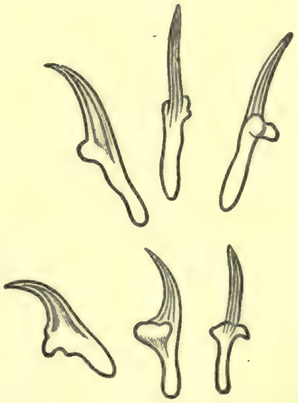


FIG. 23.

Large hook from the inner, and small hook from the outer circle of this specimen, as viewed from different sides. (Still more powerfully magnified.)

There are two ways in which we may prove that

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[1] *Krabbe*, quoted by *Leuckart*, I. p. 745.

[2] *Gerlach*, Hannover. Jahrb. 1869, p. 69. *Roloff*, Mittheilung. aus der thierärztl. Praxis in Preussen. 16, p. 167, 1869.

[3] *Leisering*, Jahresbericht. d. ges. Med. 1870, I. 495.

the Cysticercus cellulosæ is the early stage of development of the Tænia solium: either by producing Cysticercus cellulosæ in the pig, by feeding it on the ripe segments of T. solium, or by breeding Tænia solium in the intestine of a man by giving him pork to eat that contains the Cysticercus cellulosæ. Now both of these methods of proof have been frequently successfully carried out, though, of course, the latter one less often than the former. The mutual relation of the two is therefore fully established.

The first person who succeeded in producing the Cysticercus cellulosæ in the pig by feeding it on segments of the T. solium containing eggs, was van Beneden,[1] and similar experiments were afterwards successfully made by Haubner and Küchenmeister,[2] Leuckart,[3] Gerlach,[4] and many others.

Küchenmeister[5] was the first who succeeded in producing Tænia solium in the human intestine out of Cysticercus cellulosæ. To a culprit condemned to death he administered a number of embryos three days before his execution, and afterwards found ten had become everted and changed into Tænia, some of which were adherent to the wall of the intestine.

In another experiment[6] the culprit was given twenty embryos four months, and the same number two and a half months, before his execution. At the post-mortem, nineteen Tæniæ were found, some of which had mature segments.

Leuckart[7] relates another successful experiment. A young man swallowed, of his own accord, four embryos. Four months afterwards he expelled mature tapeworm segments, and, after being treated, two examples of Tænia solium were expelled. Two similar experiments were also successful.[8]

I myself have to record an experiment very similar to those of Küchenmeister. A person suffering from tubercle swallowed twenty-five embryos, freshly taken from a pig, eighteen days before his death. At the post-mortem twelve worms were found in the intestine. They had all the characteristic marks of the Tænia solium, but were, however, all very small, and in none was there any formation of segments visible to the naked eye. The search was rendered so difficult—by the intestines being filled with a quantity of turbid yellow chyme—that at first I had little hope of finding the young Tænia. It is, therefore, not to be wondered at if all the animals that were introduced were not found, even supposing that every one of them had become developed.

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[1] Annales des sci. natur. 1854, I. p. 104.

[2] Magazin f. Thierheilk. 1855, p. 105, and "On Parasites," p. 76.

[3] l. c., I. p. 229.

[4] Jahresb. der Thierarzneischule zu Hannover. 1869, p. 66.

[5] Wiener med. Wochenschr. 1855, No. 1.

[6] Deutsche Klinik, 1860, No. 20.

[7] Die Blasenbandwürmer und ihre Entwickelung. Giessen, 1856, p. 53, On Parasitis, I. p. 283.

[8] *Bertolus*, Dissertat. sur les métamorpho. des céstoïdes. Thèse de Montpellier, No. 106, 1856. *Hollenbach*, Wochenschrift f. Thierheilk. II. pp. 301 and 353. (These are taken from *Leuckart*, Parasiten, I. p. 283.)

There have been numerous experiments made to try if by feeding animals on the Cysticercus, the corresponding Tænia could be produced, but in vain. Like many others, I too have made unsuccessful experiments on dogs, rabbits, and guinea-pigs, and also on a monkey, the Macacus cynomolgus from the East Indian Islands. The tape-worm heads that had been introduced into the first animals were sometimes found as late as twenty-four hours after their introduction, after which time they disappeared entirely, evidently having been digested.

Distribution.

Tænia solium is only found in the small intestine of man. The head is usually fastened to the mucous membrane of the intestine in its upper third, and takes such a firm grip that the neck often gives way before it will let go its hold. The neck and the first segment of the worm usually lie coiled up in a mass about the head of the animal. From this point the worm extends downwards through the small intestine, being folded on itself here and there, and usually reaches to its lower third, seldom as far as the cæcum. Mature segments, either single or in short pieces, are usually found about the lower portion of the colony, and, when the worm is quite fully grown, ripe segments are usually to be seen scattered singly throughout the large intestine.

Up to the present time, Tænia solium has not been found except in man; we must therefore look upon it as being peculiar to him. The many endeavors to produce it in animals have all been, as has already been mentioned, fruitless.

The above description is founded on the position in which the worm was found in post-mortems made shortly after death.

Up to the present time we do not possess thoroughly reliable accounts as to the geographical distribution of the Tænia solium, for it has not been distinguished by authors from the Tænia saginata. Since its distribution, however, must depend on that of the animal in which it exists in an embryo condition, we may conclude that it corresponds to that of the pig. Hence, Tænia solium will be found among all nations that do not despise pork.

Tænia solium is very frequent in Europe and America. It would appear not to be quite so common in the remaining three divisions of the globe. In Europe, the middle of Germany is remarkable as being the land where it is most widely diffused.

In Holstein I meet with the Tænia saginata four times as often as the Tænia solium.

Of 100 cases that Krabbe[1] observed in Copenhagen and the surrounding country, 53 were Tænia solium and 37 Tænia saginata.

Tænia Saginata.[2]

The Unarmed or Fat Tape-worm.

The Tænia saginata, when fully developed, is far larger, and is in its general formation much stronger, thicker, fatter, and less transparent than the Tænia solium, and grows to a length of six metres. The head is far larger than that of the Tænia solium, measuring 2.50 mm. broad. It is furnished with four powerful sucking-discs, but the rostellum and circlet of hooks are wanting, and in place of the latter, there is in the centre of the head a

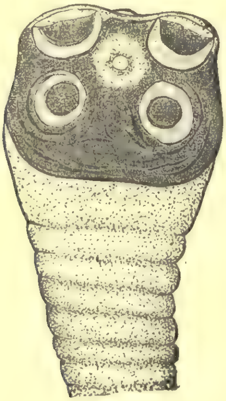

FIG. 24.

Strongly pigmented head of T. saginata.

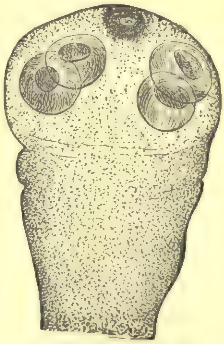

FIG. 25.

Head of T. saginata without pigment.

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[1] Ugeskrift for Læger. 3 R. VII. No. 7, 1869.

[2] Concerning the name, vide p. 687.

small frontal-sucker. At times it is quite free from pigment, at others it is more or less pigmented, while occasionally it is almost saturated with black pigment, the coloring matter being especially massed round the suckers.

Leuckart says that there are small rudimentary hooks round the frontal-sucker.

The neck is very short, and a few millimetres behind the head we can readily make out with the naked eye the indications of commencing segmentation.

The ripe segments average, when in a relaxed condition, from sixteen millimetres long and seven mm. broad to twenty mm. long and five mm. broad. The genital pores vary irregularly from one side to the other, and are situated on the margins of the segments and somewhat behind the median line.

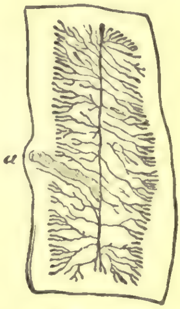

Fig. 26 (10).
Ripe segment of Tænia saginata. (Magnified six diameters.)

What was said before about the measurements of Tænia solium (v. p. 707) is equally true about those of Tænia saginata.

The uterus has on each side from fifteen to twenty lateral branches, which divide dichotomously. In other respects the sexual apparatus closely resembles that of the Tænia solium.

The eggs are usually somewhat oval, and also somewhat larger than that of the Tænia solium; they measure on an average 0.035 mm. broad and 0.039 mm. long. Their structure is quite the same as that of the eggs of T. solium, but the shells are slightly thicker. The enclosed embryo consists of a mass of protoplasm with three pairs of small chitine-hooks.

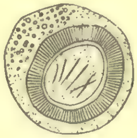

Fig. 27.
Egg of T. saginata, with six-hooked embryo.

The size and shape of the eggs do not enable us to distinguish them from those of T. solium. For the eggs of the latter worm, when seen from the side, are somewhat oval, and the difference in size is so small that it could only be appreciated when the eggs of the two species were placed together. The eggs, too, of the same species vary greatly in size. The result of very numerous measurements of eggs gave 0.047 mm. in length and 0.043 mm. in breadth, as a very rare maximum, the average minimum being 0.037 and 0.032 millimetres. In estimating the average size given above, this exceptional maximum was not included. Eggs measuring 0.041 mm. long and 0.036 mm. broad were not unfrequently met with, and are included in the above average.

The variations in the size of the eggs of Tænia solium were not nearly so great.

The thickness of the egg-shells of T. saginata is from 0.0057 to 0.0064 mm.; of Tænia sol., from 0.0050 to 0.0057 mm.

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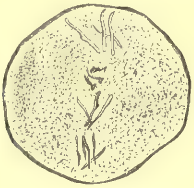

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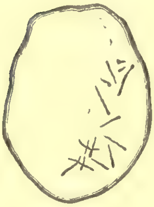

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Figs. 28 and 29.
Embryos of T. saginata, with numerous hooks.

Fig. 30.
Hooks largely magnified.

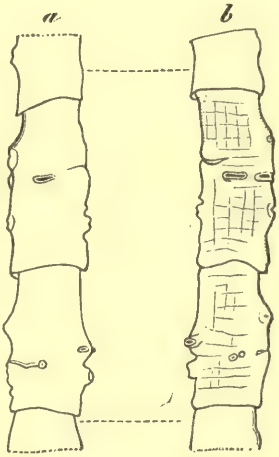

Fig. 31.
Deformed segments of T. saginata, with numerous genital pores, some of which are situated on the surface of the segments.

It is not at all uncommon to find embryos of T. saginata that have numerous hooks. In one example of T. saginata, which was in other respects deformed, I found numerous embryos with twelve and sixteen hooks, and one that had as many as thirty-two. Some of these hooks were perfectly formed; others were nothing but short, clumsy rods of chitine.

Deformities similar to those met with in the T. solium are of frequent occurrence. Very often the segments are stunted, or sterile, or full of flaws. At other times several are joined together, and form one immense segment, having several genital pores. If several such segments are found, it is usual for some of the genital openings to be situated on the broad surface of the segment, instead of on the margins. Thus, in each of the cases represented in the accompanying wood-cut there are three genital openings on the broad surfaces of the segments.

In the Museum of the College of Surgeons in London there is a specimen of such a segment which is of gigantic size. I counted twenty genital openings on it, two of which were situated on the broad surface of the segment. The other side of the segment could not be seen.

Double monstrosities, similar to those found in T. solium, also occur, and are, it would appear, more common. Thus, the worm described by Küchenmeister[1] as occurring especially among the Hottentots, as well as those described by Bremser, Cobbold, and Cullingworth, belongs most probably to this species. None of the heads of these worms, however, have as yet been obtained.

The ruminants must be looked upon as the hosts of the T. saginata during its Cysticercus condition, the embryos having been found in the voluntary muscles, in the heart, and in the brain of these animals. Up to the present time they have only been found in the cow and in the giraffe; but they have been artificially produced in the calf, the goat, and the sheep, by feeding these animals with segments of the worm. This is probably because the ruminants, taken as a class, present conditions that are favorable to the development of the Cysticercus of the T. saginata.

It is a most remarkable fact, that, though T. saginatæ are so common, their embryos have very rarely come under observation. With the exception of Knoch,[2] who, writing from St. Petersburg, speaks of them as of common occurrence in cow's flesh, no one has observed them in Europe, except Professor Möbius,[3] who found them in a giraffe that was in the Zoological Gardens in Hamburg. The Pathological Institution of Kiel has in its possession a portion of an ox-tongue containing them, which was presented to it by Dr. Closs, of Frankfort-on-the-Main. In the Senkenberger Museum there is another portion of this tongue. The only other person who, so far as I know, has seen them is an English doctor, who met with them in India, and who says they are constantly met with in the flesh of the cow, and who, without recognizing their true nature, has given a most accurate description and drawing of them.[4]

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[1] For the references, see under T. solium, p. 710.

[2] Petersb. med. Zeitng. X. p. 245, 1866.

[3] *Noll*, Zoolog. Garten. XII. p. 162.

[4] In a report of the Indian Government for 1770, which I had the opportunity of seeing at the house of Dr. Cobbold, in London.

The Cysticercus of the T. saginata has been bred in *cows* by Leuckart,[1] Mosler,[2] St. Cyr,[3] Cobbold,[4] Zürn,[5] and Probstmayr.[6]

In the *goat* by Zenker[7] three times, and by me once.

In the *sheep*, once by me, and probably once by Cobbold.[8]

Unsuccessful attempts have been made to produce them in the *sheep*, by Zürn and Leuckart;[1] in *swine*, by Küchenmeister, Zenker,[7] Leuckart,[1] and Schmidt;[1] in the *dog*, by Probstmayr[6] and by me; in *rabbits*, *guinea-pigs*, and an *ape*, by me.

The particulars of my experiments will be given in another place.

There are many grounds for the striking fact that the Cysticercus of this very prevalent species of Tænia should be met with so very rarely. One of the principal of them is, that the conditions are not favorable to the introduction of a large number of eggs into the cow; for the T. saginata deposits the greater number of its eggs while still in the intestine. These are passed along with the fæces, to which, from the habit of the animal, it seldom has access. Further, the segments, from which most of the eggs have escaped, are being continually expelled singly, and are consequently distributed over a very large area. Thus, even if one of them is accidentally introduced into the animal along with its fodder, no very extensive invasion of embryos can take place; and, finally, the embryos, being distributed over such a large animal, are not so readily found.

The Cysticercus of the T. saginata consists of an oval cyst, which, when developed, is perhaps somewhat smaller than that of the T. solium. At one end of the cyst may be seen the place into which the head, which is inverted like the finger of a glove, is inserted. It is separated from the rest of the contents of the cyst by a delicate membrane, which is known as the "mantle." The head exhibits the marks characteristic of the Tænia saginata, viz., four powerful sucking-discs, without any hooks. The worm is fully grown twelve weeks after the animal experimented on has swallowed the eggs, though the embryos continue developing for some time longer.

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[1] Parasiten. I. pp. 296, 406, 414 (in sheep), 747 (in swine).

[2] Helminthol. Studien. Berlin, 1864.

[3] Journal de l'anatomie, 1873.

[4] Supplement to the treatise on Entozoa, p. 18.

[5] Zoopatholog. Untersuchung. 1872, pp. 52 and 57.

[6] Jahrb. d. Münchener Thierarzneischule, 1869–1870.

[7] Sitzungsberichte d. Erlanger phys.-medic. Societ. 1865-67, p. 15; and ibid. IV. p. 71, 1872.

[8] Med. Times and Gaz. June 15, 1866.

The embryo (at least in the sheep and goat) is surrounded by a very thick capsule composed of connective tissue, the inner surface of which is usually covered with a copious, yellow, unctuous deposit.

The Cysticercus of the Tænia saginata seems to be much shorter-lived than most of the other Cysticerci, numbers of them dying even before they have become fully developed, and a large portion perishing prematurely after being fully developed. After the death of the embryo, it and its capsule are transformed into a small node filled with a greasy, greenish-yellow pulp.

With regard to the identification of the T. saginata, there can nowadays no longer be any doubt but that Küchenmeister has correctly laid down its distinguishing characteristics. I myself have had the opportunity of examining thirty perfect specimens of the T. saginata, two while still in the small intestine. In every one of these cases the characteristic features by which it is distinguished from the T. solium were well marked; the head was much larger and the sucking discs much more powerful; the rostellum and circlet of hooks were absent; the number of the lateral branches to the uterus were much more numerous, and the branches themselves divided dichotomously; the whole worm was fatter and more powerful.

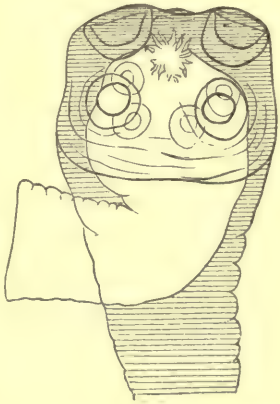

FIG. 32.
Head of the T. solium within that of the Tænia saginata showing the difference in their size. Drawn with a prism.

In the rare case of both T. solium and T. saginata being present at the same time, difficulty might be experienced in making a positive diagnosis till after the expulsion of the worms.

It is no longer necessary to refute the idea that T. saginata is nothing but an individual T. solium, which has lost its hooks and rostellum, and whose sucking-discs have become hypertrophied from the increased amount of work thrown upon them. Examples of Tænia solium are frequently observed which possess very few hooks, and whose heads and suckers differ neither in size nor in appearance from those of worms having the full number of hooks. I have in my possession such a head, the smaller circlet of hooks alone being present.

The successful results that have attended the attempts to produce the embryos in ruminants by feeding them upon the eggs, coupled with the lack of success of all attempts to produce them in other animals, especially in swine, are incontrovertible proofs that they do not belong to the same species.

The doubts also that have been raised as to the Cysticercus cellulosæ of the pig being the youthful form of the T. solium, have been quite silenced by the discovery of Küchenmeister's, that there are two varieties of the Tænia. Before this it had been especially objected that tape-worms were very frequently met with in persons who entirely abstained from pork as an article of diet.

Distribution.

The T. saginata inhabits the small intestine of man. The head is attached in the neighborhood of the termination of the upper third of the intestines, and is surrounded by coils of the upper portion of the colony. From this the worm extends downwards in loose coils, seldom reaching as far as the cæcum. Mature detached segments are found about the lower portion of the worm. It is peculiar to man, and has never yet been found in one of the lower animals.

As to the geographical distribution of the T. saginata, what has been said on the subject when speaking of the T. solium holds good in a general way here. From the accounts, however, that have reached us, we can readily see that it is much more common, and is met with over an immensely greater area, than the T. solium.

The countries where it is most prevalent are Africa and Asia. In Abyssinia both old and young, men and women, suffer, and travellers who adopt the Abyssinian customs are certain to acquire them. In the rest of Africa they appear to be quite as frequent.

In Europe the worm is widely spread, though not in such enormous numbers as in Africa. It is the worm most commonly met with in Southern, and part of

Northern Germany, while in the Central and North-western parts T. solium exceeds it in frequency.

Of one hundred cases of tape-worm that Krabbe (l. c.) observed in Copenhagen and the surrounding country, thirty-seven were cases of T. saginata.

In Vienna, Bremser and Wawruch only observed the T. saginata.

Tænia Elliptica (Cucumerina).

This is a delicate tape-worm, which measures from one hundred to three hundred millimetres in length. The head is furnished with a rostellum, which the animal has the power of projecting forwards, on which are placed about sixty hooks, arranged irregularly in three or four rows. The anterior portion of the body is like a thread, and the segments are very short; more posteriorly they become somewhat longer. As they become ripe, the divisions between the segments become more and more marked, so that the worm presents a chain-like appearance. The ripest segments have a reddish-white color, and very readily become detached. They creep about actively in the intestine, and are then either expelled with the fæces or escape of themselves. The eggs measure 0.05 mm.

It is doubtful whether two different species inhabit the dog and the cat, for the differences that have been given are very slight, and, it would also appear, not always present. In each segment there is a double set of sexual organs, and there is a genital pore situated on each margin of the segment.

According to Melnikow,[1] the embryo of this tape-worm inhabits the dog louse, Trichodectes canis.

This worm inhabits, in considerable numbers, the small intestine of the dog and cat, and has frequently been found in man, especially in children.[2]

Tænia Flavopunctata.

This is a small tape-worm, measuring from 200 to 300 mm. in length. Up to the present time the head has not been seen. The

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[1] Arch. f. Naturgeschichte, 1869.

[2] *Linné*, Amoenitat. acad. II. p. 81. *Salzmann*, Deutsche Klinik, 1861, 32. *Leuckart*, l. c., p. 402. *Krabbe*, l. c.

segments measure in the forepart of the body from 0.2 to 0.5 mm. in length, by from 1 to 1.25 mm. in breadth. In the median line, but more towards the posterior edge of each segment, is a tolerably large yellow spot, which is the receptaculum full of seed. Towards the free end of the worm, the segments increase to 1 mm. long, and from 2 to 2.3 mm. broad. Instead of a yellow spot, the whole segment is now of a brownish gray color, from being almost entirely filled with eggs. The eggs, which have a smooth, double covering, measure 0.06 mm. The uterus is simply a smooth cavity that occupies nearly the whole segment. All the genital pores are situated on the same margin.

This worm has only been found once. Weinland[1] found fragments of six specimens, that had been expelled by a child nineteen months old, in the Museum of Pathological Anatomy in Boston.

Tænia Nana.

A tape-worm 15 millimetres in length. The head, which is spherical, has four round sucking-discs, and an oval rostellum, with a single row of very small hooks. The segments, which are from 150 to 170 in number, are short, the broadest of them measuring 0.5 mm. The uterus, which corresponds to the shape of the segments, contains numerous eggs, which have a double envelope, and measure 0.04 mm. in diameter. The genital pores are all situated on the same margin.

This small tape-worm was once found by Bilharz[2] in Egypt. It occurred in innumerable numbers in the intestines of a boy who had died of meningitis.

Spooner[3] states that he has found it in a young man, in whom it gave rise to symptoms similar to those produced by the other varieties of Tænia.

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[1] Beschreibung zweier neuer Tänioiden aus dem Menschen. Jena, 1861. p. 8. Reprint from the Transactions d. k. L. C. D. A. XXVIII.

[2] Zeitschrift. f. wissenchaftl. Zoolog. 4, p. 64, 853. *Leuckart*, l. c., p. 393.

[3] Am. Journ. of Med. Sci. Jan. 1873. (Jahresbericht, 1873, II. p. 6.) The original was, unfortunately, not at my disposal. Considering the great similarity between the T. nana and the T. flavopunctata, we may be allowed the conjecture that the worm probably belonged to the latter species, especially as Spooner's observation was made in America. It is to be regretted that nothing is said about the formation of the head.

Tænia Madagascariensis.

This worm has, up to the present time, only been met with broken up into fragments ; the head has not as yet been found. The segments in the middle of the worm measure 0.8 mm. long, and 2.2 mm. broad ; those more posteriorly measure from 3 to 4 mm. The genital pores are all on the same margin. The eggs are said to be packed up in capsules, each containing from 300 to 400 eggs. Of these capsules there are from 120 to 150 in each segment.

This worm was observed twice by Grenet in Mayotta (Comoro Islands), and has been described by Davaine.[1] Grenet saw it in two children, one of whom was eighteen months and the other two years old.

Bothriocephalus latus.

Bothriocephalus latus is the largest of all the tape-worms that inhabit the human intestines, and is said to attain to a length of from 5 to 8 metres. The head is almond-shaped, and about 2 mm. long and 1 mm. broad. The flat surfaces of the head correspond to those of the body. Along each side of the head runs a fissure-

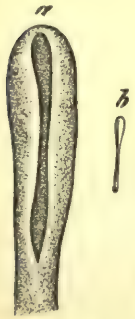

FIG. 33.

a, Head of Bothriocephalus latus seen from the side (magnified). *b*, The same seen from above (natural size).

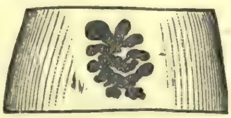

FIG. 34.

Ripe segment of Bothrioceph. lat. (mag. 6 diam.).

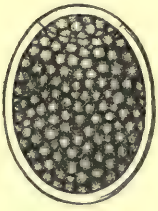

FIG. 35 (5).

Young egg of the Both. latus.

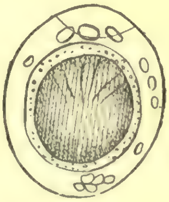

FIG. 36.

Egg in which the embryo has become developed (Leuckart).

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[1] Archives de méd. nav. 1870. (Nouv. dict. de méd. et de chir. red. Jaccoud, 13, p. 371, 1870.)

like pit in which its suction apparatus is placed. When fresh the worm has a dull bluish gray color.

The undeveloped *segments* are from three to four times as broad as they are long, or from 10 to 15 mm. broad and from 3 to 4 mm. long; the ripe ones are almost square, and measure about 5 mm. each way.

The *genital pores* are situated in the centre of the broad surface of the segments, and are all on the same side. The segments that are nearly mature show a brown marking, like a rosette, in the centre. This is caused by the numerous convolutions of the uterus, which, though at first a straight canal running from before backwards, has assumed this shape and color from the number of brownly pigmented eggs that it contains.

The *eggs* are oval, with thin shells, which are of a light brown color. They are furnished at one end with an operculum or lid, which is separated from the rest of the egg by a well-marked line, and is very easily recognized. They contain a more or less coarsely granular mass. If the ripe eggs are preserved in water, in the course of some months six-hooked embryos become developed in them, which creep out as soon as the lid has been cast off. These embryos are surrounded by an envelope which is set all over with long cilia. By means of this covering—the cilia of which are in active motion—they swim about in the water. After from four to six days the embryo puts off this covering, but it is still enveloped in a clear albuminous mass.

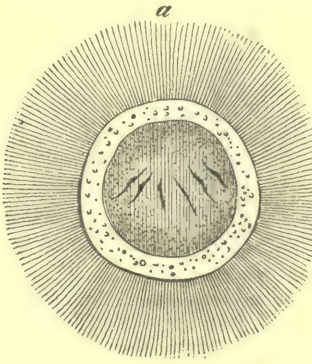

FIG 37.
Embryo with ciliary envelope (Leuckart).

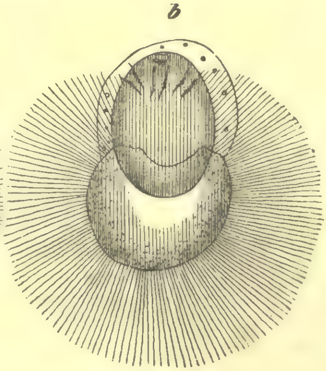

FIG. 38.
Embryo casting off its ciliary envelope (Leuckart).

This, too, it loses after a time, without however at once dying. Leuckart has seen them creeping about for some time afterwards by means of amœboid-like contractions. Their further fate is wholly unknown.

Böttcher thinks that the broad surface of the head corresponds to the edge of the segments, which is exactly the opposite to Leuckart's idea (Virchow's Archiv, 30, p. 103, 1864).

Knoch,[1] of St. Petersburg, is of opinion that eggs of the Bothriocephalus latus, if introduced into the intestinal tract of man, immediately begin to develop into tape-worms. He supports this opinion by a number of unsuccessful attempts he has made to procure the animal in the intermediate condition, by bringing the embryos of Bothriocephalus into contact with different aquatic animals. In no case was there an immigration of the embryos nor the production of any intermediate stage. On the other hand, after having fed two dogs, one several times with the undeveloped eggs, and the other for eight days with embryos, he found in the former, four months afterwards, seven examples of Bothriocephalus in different stages of development, and six weeks afterwards he found in the latter two worms measuring one and one and a half inches, and also two mature worms measuring eighteen and twenty-one inches respectively. In another dog, which a year previously had been given embryos, and two months previously a piece of a Bothriocephalus, he found a young Bothriocephalus in the small intestine, and a fully grown one further down. Knoch thinks that these worms were the products of the embryos he himself gave the dogs, and therefore considers himself justified in holding the above opinion.

Since, however, Bothriocephalus is very often found in dogs (see further on), these experiments prove very little, especially as similar experiments have failed when made in places where Bothriocephalus was not prevalent.[2]

Therefore, until similar experiments to those made by Knoch have been made successfully in some places where Bothriocephalus is not prevalent, we must continue to doubt the accuracy of the conclusions he has drawn from them.

It is far more probable that Bothriocephalus latus exists during its immediate stage of development in some aquatic animal, who acts as its temporary host. This view is supported by the frequent occurrence in fishes of the encapsuled cestode worms of Bothriocephalus, and also by the fact, authenticated by Abildgard, that the Bothriocephalus solidus, which occurs in the abdomen of fishes, only becomes developed into a tape-worm after its host has been devoured by some predaceous bird, in the intestines of which it becomes developed.

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[1] Mém. de l'acad. etc. de St. Petersbourg. VII. Sér. V. No. 5, 1862. Virchow's Arch. 24, p. 453, 1862. Journ. de l'anatomie, etc. 1870, p. 1. For the minute structure compare *Böttcher*, Virch. Archiv, 30, p. 97, 1864. *Sommer* and *Landois*, Zeitsch. f. wissensch. Zoolog. XX. 1872.

[2] Leuckart, I. p. 764.

Distribution.

Bothriocephalus latus exists in the intestine of man usually single, but now and then several examples occur together. In rare cases it occurs in company with the two varieties of Tænia. It is, however, not confined to man, but has been frequently met with in dogs.[1]

The geographical distribution of Bothriocephalus lat. is very limited, for with very few exceptions it does not occur beyond fixed bounds, and we have at present no authentic cases where it was met with outside Europe. In Europe its chief seat is on the north-east coast of Sweden, in Finland, St. Petersburg, the Baltic provinces, and Poland, then in the western parts of Switzerland with the neighboring parts of France, and more rarely in Belgium and Holland, in the eastern provinces of Prussia and in Pomerania, also in Zeeland.

In Haparanda there is said hardly to be a house in which one or more persons do not possess Bothriocephalus.

According to Odier, one-fourth of the inhabitants of Geneva suffer from it.

Cruse[2] found it in six per cent. of four hundred and eighty-two post-mortems made in Dorpat.

In one hundred cases of tape-worm, observed by Krabbe,[3] in Copenhagen, it occurred nine times, and all these patients came from Zeeland.

It is said that fifteen per cent. of the inhabitants of St. Petersburg suffer from Bothriocephalus.[4]

In other regions it is only met with in isolated cases, and is then usually imported. Thus single cases are recorded as occurring in Rhenish Hesse, Würtemberg, Hamburg, London, St. Malo, Montpellier, Rome, and Zurich. One case was also met with in Holstein, and the worm is to be found in the collection of the Pathological Institution in Kiel.

All the accounts we have of Bothriocephalus latus occurring in countries out of Europe are due to confounding this worm with the T. saginata. Thus Java (Schmidtmüller), Ceylon, the coast of Natal in South Africa (Fritsch, Archiv f. Anatom. u. Phys. 1867, p. 733), have been mentioned; in the latter place the use of cow's meat is given as the reason why this worm is so prevalent.

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[1] *Leuckart*, l. c., II. p. 422. *Krabbe*, Ugeskrift for Læger. 3. R. VII. No. 7, 1869, met with Bothr. lat. twice in the dog.

[2] Dorpat. med. Zeitschr. II. p. 315.

[3] Ugeskr. for Læger, 3. R. VII. No. 7, 1869 (Jahrb).

[4] Compare *Hirsch*, Historisch-geographische Patholog. II. p. 294.

The accounts of the frequent occurrence of this worm in Thüringen[1] appear to be founded on the same mistake.

Bothriocephalus Cordatus.

A second worm, having suction-pits in place of suction-discs, has been described by Leuckart[2] under the name Bothriocephalus cordatus. It is smaller than the last-described variety, sometimes by as much as one hundred and fifteen centimetres, and has a short, broad, heart-shaped head, the suction-pits on which correspond to the flat surfaces of the worm, and not, as in Bothriocephalus latus, to its margins. The body commences immediately behind the head, the segment increasing very rapidly in breadth, so that the anterior part of the worm has the shape of a lancet. At a very short distance, about three centimetres from the head, the segments contain fully developed sexual organs, and at a distance of another three centimetres they attain their maximum size, and measure from seven to eight mm. in breadth. They are of a dark gray color round the margin, but are bright in the centre. Numerous calcareous corpuscles are scattered throughout the parenchyma of the body. The rosette formed by the uterus is smaller and more elongated than in Bothriocephalus latus, and has more numerous (6–8) offshoots. The eggs are somewhat larger than those of the Bothriocephalus latus, but are in other respects very similar to them.

Nothing is known about the development of this worm.

The Bothriocephalus cordatus is found in Greenland, where it frequently infests dogs. It has once been met with in a pregnant woman. The symptoms that were present in this case are clearly referable to the pregnancy.

Böttger[3] denies that the position of the head is a distinguishing characteristic of this worm, as has been stated by Leuckart. He holds that the suction-pits of the Bothriocephalus latus correspond to the flat surfaces of the segment, and not to their margins, and further that individual worms of this species (Bothriocephalus latus) differ greatly from each other in their general appearance. He therefore negatives the supposition of there being two distinct species. The only thing that was peculiar about Bothriocephalus cordatus was the large number of calcareous corpuscles observed in them by Leuckart.

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[1] *Conta*, l. c.

[2] l. c. I. p. 437.

[3] Virchow's Archiv, 30, pp. 103 and 146, 1864.

Suctorial-Worms.

(Trematoda, Saugwürmer.)

Distomum Crassum.

This is a thickish, leaf-shaped leech, measuring from four to six ctm. in length, and from 1.7 to two ctm. in breadth. Its skin is smooth and free from spines. At the more pointed extremity are situated two round sucking-discs at a distance of three mm. from each other. At the anterior edge of the more posterior and larger of them is situated the sexual opening. The intestinal canal is double, and does not branch. The uterine convolutions are confined to the anterior part of the body. The testicles are two plump, lobed organs, situated in the centre of the posterior half of the animal.

This worm has only been met with in man. Busk,[1] in 1843, found fourteen examples of it in the duodenum of a Lasker who died on board one of the Thames hospital ships. The disease the man died of does not appear to have depended in any way on the presence of the worm.

Distomum Heterophyes.

This worm has a pear-shaped body, from 1 to 1.5 mm. long, and 0.7 mm. broad, which is pointed in front and rounded off behind. On the anterior portion of the body the skin is thickly set with spines. Both the frontal and abdominal suckers are well developed; the latter is the larger, and is situated in the centre of the animal's body. The pharynx, which lies a little posterior to the frontal sucker, divides into two branches just in front of the abdominal sucker, these two branches extending through the rest of the animal's body. The genital opening is situated behind and a little to the side of the abdominal sucker ; behind it are the brown-colored convolutions of the uterus—indeed the whole animal has a red color imparted to it by the reddish-brown color of the eggs.

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[1] *Davaine*, l. c. p. 254. *Cobbold*, Linn. Soc. Proceed. 1861.

This diminutive leech has been found twice by Bilharz[1] in Egypt, in the small intestines, one of the patients being a boy. The animals were so numerous that Bilharz collected several hundreds without any difficulty, and still left the chief portion behind.

It is not known whether this worm is of any medical importance.

Round-Worms.[2]

Ascaris Lumbricoides. Der Spulwurm.

This is a cylindrical worm, having a light-brownish or dirty reddish-yellow color, which in rare cases approaches to red. It tapers towards both extremities,—more quickly, however, posteriorly than anteriorly, though not to the same extent. On the anterior extremity are placed three semicircular lips, the bases of which are separated from the remainder of the body by a well-marked circular furrow. The opening for the mouth is situated between these three lips or papillæ, and is continuous with the œsophagus, which is from six to eight mm. in length. To this the intestine is joined, which is very long and of a slight brownish or greenish color, the anus being situated at a short distance from the posterior extremity of the animal.

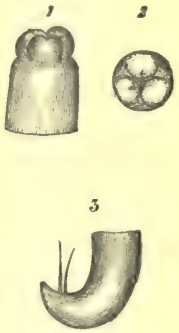

FIG. 39.
Ascaris lumbricoides. 1. Head seen from the side. 2. Head seen from above. 3. Tail of the male worm.

The *male*, when first taken from the intestine, measures eighteen ctm., but has the power of stretching to twenty-seven ctm. Its tail is always bent on the abdomen like a hook. The testicle appears like a single white thread which has numerous convolutions, and opens into the club-shaped spermatic duct. This latter opens into the end of the intestine, and with it forms a cloaca, from the orifice of which the two slender spicules may often be seen projecting.

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[1] Zeitschrift f. wissenschaft. Zoolog. 4, p. 62, 1853.

[2] The last part of *Leuckart's* excellent work, "Die Menschlichen Parasiten," did not appear till after the MS. of this chapter had been sent to the editor, and could not, therefore, be made use of.

The *female* is twenty-five ctm. long, but can stretch up to thirty-two ctm. The female sexual organs consist of two very long, white, thread-like ovaries, which pass gradually into the two lateral branches of the uterus. The two ovaries of a female, which was one hundred and forty-three mm. long, measured fourteen hundred mm. The two uterine branches unite and pass into the vagina, which measures about six mm. in length, and opens externally at the termination of the anterior third of the body.

The *eggs*, the total number of which in the sexual organs of a female has been estimated by Eschricht and Leuckart at about sixty millions, are, when unripe, conical and are adherent to a longitudinal band. When ripe, they are oval, and have a dark, tough, double shell, and very dark granular contents. They measure from 0.05 to 0.06 mm. in diameter. The ripe eggs that are expelled into the intestine are always surrounded with an albuminous coating, the surface of which is covered with small irregular elevations, and this is usually of a dark brownish-green color from the effects of the gall.

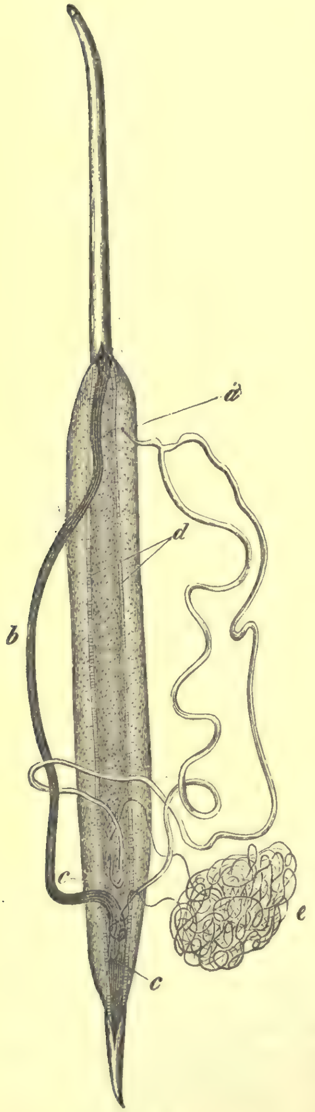

FIG. 40.

Female Ascaris lumbricoides, 143 mm. long. *a*, Vagina. *b*, Intestine. *c*, Junction of uterus and oviduct. *d*, Longitudinal bands. *e*, Coils of the ovary and oviduct.

As a rule, the eggs, which are often expelled in countless numbers along with the fæces, when left in a moist place do not usually develop any further for several months, sometimes not till after an indefinite period; gradually, by the usual process of cleavage, a long, mulberry-like body is formed, which becomes thinner and more elongated, and gradually develops into a worm-like embryo, from 0.3 to 0.4 mm. long. The eggs have great power of retaining their vitality in spite of external influences. Thus repeated freezing or desiccation did not prevent their subsequent development. They do not lose their power of development for years, and the young embryo, while in the shell, also retains its vitality for years.

We are still quite in the dark as to the further fate of the embryos or as to the means by which they obtain entrance into the intestines of man. Three ways are possible: the first is, that the eggs containing the embryos are conveyed directly into the stomach of some human being; from the action of the gastric juice the embryos are then set free, and, after casting their skins once or twice, arrive at their final form. The second possibility is, that the eggs are swallowed by some other animal, in which the embryos become free and pass through their intermediate stage, and finally, in some way, are introduced into man and become further developed. Or, lastly, the embryos leave the eggs without these having ever come into the stomach of an animal, then exist for some time as independent animals, and finally, after being introduced into the human stomach, attain their full development.

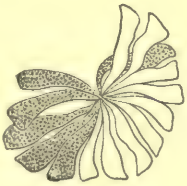

FIG. 41.

Unripe eggs of Ascaris lumb.

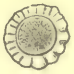

FIG. 42.

Ripe egg of Ascaris lumb. with albuminous envelope.

The last of these possible ways is the most improbable, and the hardness of the shell of the eggs is also against it. Above all, however, no one has ever seen the embryos escaping from the eggs, nor found them free, although eggs have been watched with the greatest attention for years by numerous observers. The formation, too, of the embryo speaks against it, for it is provided with a tooth-like appendage on its head similar to that possessed by other embryos which are not destined to lead an independent existence.

The two first suppositions are probable; but which is actually the true one must, for the present, still be left undecided.

Numerous experiments have been made in order to determine this question, but up to the present time without result.

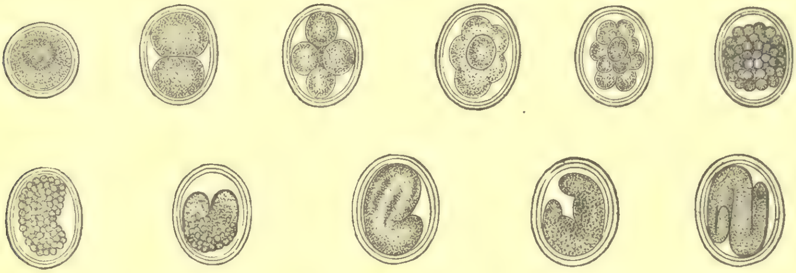

FIG. 43.
Development of the egg of Ascaris lumbricoides.

Thus Leuckart (l. c., II. p. 221) has fed dogs, rabbits, pigs, and mice on the ripe eggs of Ascaris lumbricoides without result; the eggs either passed through the animal unchanged, or were thoroughly digested. Leuckart has himself made, and caused to be made by others, a series of similar experiments on men, which all gave a negative result,[1] and his experiments of feeding horses with the eggs of Ascaris megalocephala, and cats and dogs with those of Ascaris mystax, were equally fruitless.

Davaine also has made similar fruitless experiments on cows, in which, otherwise, the Ascaris lumbricoides is often found.

It was only in the lower part of the small intestine of the rat that Davaine once saw some of the embryos free, some still in the shells, and some in the act of creeping out of them. He also found the embryos free and moving about in the fæces of a rat which had for a long period been fed on milk containing fully matured eggs which were five years old. He also introduced eggs (enclosed in small glasses covered with linen) containing embryos into the intestines of a dog, and afterwards found that the eggs that were not quite fully developed still remained, but the ones that were fully developed had disappeared, and, instead of them, there were several free embryos.

Leuckart has made experiments on numbers of the smaller animals, in order to find out if they were the temporary hosts of the embryos. They all, with the exception of the water-palmer (Wasserassel), rejected the eggs; it however passed them unharmed and still containing the living embryos.

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[1] *Mosler*, Virch. Arch. 18, p. 249.

One thing is certain, that, whether an intermediate host be necessary for their development or not, they cannot have made much progress before they are introduced into the alimentary canal of man; it is, on the contrary, probable that the young ones, when so introduced, still possess the embryonal form. Their further development must take place very rapidly, for young ones only 2.75 mm. long already have the three semi-circular lips as fully developed as a full-grown worm. While still in the egg the embryo casts its skin and obtains a tooth-like appendage at its anterior extremity. By further casting of its skin its final form is arrived at.

The smallest examples of Ascaris lumb. as yet known were found by me in the small intestine of an insane patient, fifty-two years of age.[1] They were eighteen in number, and were all delicate, slender worms, measuring from 2.75 to 13 mm. The characteristic form of the head was well marked, but it was still impossible with any certainty to distinguish the sex.

Leuckart found in the stomach of a cat numerous embryonal round-worms, measuring 0.4 mm. in length, and beside them all the intermediate steps up to the larger examples found in the small intestine. They remain in the stomach till they have attained a length of from 1.5 to 2 mm., and then pass into the small intestine. When they have attained a length of 2.8 mm., they cast their skins and change the tooth-like boring apparatus for the three characteristic semicircular lips. These observations on Ascaris mystax make it also probable that Ascaris lumb. is introduced into the human stomach as an embryo, or not much larger.

When making post-mortems I have frequently found embryos as small as the ones already mentioned.

According to Unterberger[2] the immigration of Ascaris maculosa into the dove takes place without the intervention of an intermediate host. He fed doves, which had been proved by an examination of the fæces to be free from worms, on fæces that contained eggs, and seventeen days after they were so fed he found eggs in the fæces. No transmission took place with eggs that had been taken from the oviducts.

It is only in localities where the worm is not only very prevalent in men, but is also often found in great numbers, that there is any prospect of obtaining information as to the previous fate of the worm; for only in such places are the young forms found in sufficient quantities to afford plenty of material for making such experiments. Most of the experiments up to the present time have been made with eggs taken from the worms themselves. There would certainly be more prospect of success if a sufficient number of eggs for the purpose could be obtained from the intestines, for then alone would the conditions be truly normal. The necessary quantity could readily be procured in lunatic asylums and orphanages by treating the fæces with water and then decanting it off.

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[1] *Heller*, Sitzungsb. der phys.-med. Societät zu Erlangen. 4, p. 71, 1872.

[2] Oesterr. Vierteljahrschr. f. Thierheilk. 30, p. 38, 1868.

The development of the worm, once it has been introduced into the digestive canal, must be very rapid; for it is comparatively seldom, even when great attention is directed to it, that very young animals are found when making post-mortems. During their further development the posterior half of the body increases in size much more rapidly than the anterior. This is due to the development of the sexual organs.

In the embryo the length of the œsophagus is to the length of the whole animal as 1 : 3; in animals from 2.75 to 4.1 mm. long, as 1 : 6; in those from 4.4 to 8 mm., as 1 : 7; in those from 8.18 to 11.94 mm., as 1 : 8; in those of 13 mm., as 1 : 9; in a male specimen measuring 22.8 mm., that was expelled along with a tape-worm, as 1 : 13; in a female that measured 143 mm. (Fig. 40) as 1 : 24; and in the fully grown animal as 1 : 40.

Although in a large number of cases the whole contents of the intestine of children have, after being thinned with salt water, been searched through most carefully for young worms, only in a comparatively small number of cases have we here succeeded in finding animals as small or nearly so small as the ones before mentioned. As most of the children had died of disease, we may perhaps assume that, owing to the disease, the conditions were less favorable for their introduction, and that the worms that had been there previously had either been expelled, as we not unfrequently notice during disease, or had had time to grow large.

Distribution.

The common round-worm inhabits the middle portion of the small intestine of man, more than one example being usually present. It is seldom found in the large intestine, and is then always dead and on its way to be expelled. In the small intestine it is usually stretched out straight, or has at most a slight curve. Large entangled masses of worms are met with very rarely, and that only after death. Occasionally it makes its way by mistake to other parts of the body, as, for instance, the stomach, the œsophagus, and the larynx, and more rarely into the gall-duct, the pancreatic or the nasal duct. Under peculiar circumstances it may even be found in places that have no natural communication with the intestine.

Besides being found in man, this worm is very frequently met with in the pig and in the cow.

Many persons still doubt whether the worm found in pigs is the same as that which is found in man. Leuckart and Schneider[1] maintain that it is.

The round-worm is met with over almost the whole of the inhabited globe. It seems to be specially prevalent in warm climates and among nations that are but half civilized. Under these circumstances, also, it is much more frequently met with in large numbers in the same individual. Iceland is, according to Krabbe[2] and Finsen,[3] the only place where it is not to be found.

Among civilized nations the cultivated and well-to-do portion of the community, with the exception of the children, seem to suffer but little from this worm. The lower classes are, however, greatly afflicted, sex or age seeming to have no influence.

We have numerous accounts about the prevalence of round-worms in places that are most distant from each other. Thus, according to Huss, no one is free from it in Smaland (Sweden). The same is said to be true of many parts of France. It seems to be especially prevalent, however, in warm climates. Thus Syria, India, the Indian Archipelago, Burmah and China, Egypt, Nubia, Senegambia, the islands of Bourbon and Mauritius, the Seychelles islands (especially Mahé), Newfoundland, the Antilles, Cayenne, and the Brazils, are terribly afflicted with them.

Up to the present time we have but few reliable statistics as to their frequency. They occurred in 9.1 per cent. of all post-mortems made at Dresden, in 12.9 per cent. of those made in Erlangen, and in 17.7 per cent. of those made in Kiel.

Virchow[4] found it extremely prevalent in Würtzburg.

The statistics of Kiel that were given in the introduction refer chiefly to persons of the lower classes. At the comparatively rare post-mortems of persons of the upper and well-to-do classes, the worms were very rarely seen. The number of such post-mortems was, however, too small to be of any statistical value. Hence, all post-mortems, without exception of any class, were included in the table of statistics.

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[1] Nematoden, p 37.

[2] *Leuckart*, II. p. 288.

[3] Jagttagdser angaaende Sygdomsforholdene i Island. Kopenhagen, 1874, p. 108.

[4] Archiv, 11, p. 79, 1857.

Etiology.

Since our knowledge concerning the fate of the eggs of the round-worm do not reach beyond the formation of the embryo, we are naturally quite in the dark as to how men become infested with the worm itself. We can only, from the mode of their propagation and their distribution, entertain some conjectures and imagine in a general sort of way how it takes place.

Again, since round-worms occur so universally and in such enormous numbers among nations which are but little cultivated, whereas among civilized nations they are chiefly met with among certain portions of the community—viz., in the children of the cultivated and well-to-do people, and in the lower classes—with almost the same frequency at every age, we are justified in concluding that some circumstances common to them all, tending to produce these differences, must also be favorable to the introduction of the worms. First and foremost is undoubtedly want of cleanliness or insufficient attention to it.

The introduction of the worm is most readily brought about by means of articles of food that have become contaminated either with the eggs or embryos. This is said to be especially liable to occur from the use of fruit, vegetables, salad, and other articles of diet that are eaten raw. Contamination of the drinking water with eggs out of privies has also been blamed as a cause, and indeed Mosler[1] has demonstrated their presence in such water.

We must admit that all these ways are possible ; but, nevertheless, we have not as yet attained to anything like certainty, to obtain which much more extensive and successful experiments are required.

The idea that round-worms are introduced into the body with the water used for drinking is strongly upheld by Davaine (l. c., p. 128). He says the population of Paris, who never drink any water that has not been filtered, suffer very rarely from round-worms, whereas it is most prevalent in the country part of France. He also lays stress on the frequency with which these worms are encountered in negroes, while the whites, who quench their thirst with foreign fermented drinks, tea, filtered water, etc., are much more rarely afflicted. These two facts are, however, far from proving unanswerably that the worms are introduced in the drinking water.

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[1] Virchow's Archiv, 18, p. 248, 1860.

Pathology.

Ascaris lumbricoides is found in *both sexes* and at *every age.* However, it seems to have a slight preference for women and children.

In Dresden (see p. 735) round-worms were found in 9.1 per cent. of all post-mortems. Of these 8.1 per cent. occurred in men, 9.5 per cent. in women, and 41.6 per cent. in children. In Erlangen they occurred in 12.9 per cent., of whom 11 per cent. were men, 15.7 per cent. were women, and 13.3 per cent. were children. In Kiel they occurred in 17.7 per cent., of whom 12 per cent. were men, 22.2 per cent. women, and 21.8 per cent. children.

The *seasons of the year* seem to influence the frequency of their occurrence. Many observers agree that they are most frequently met with in autumn, so that the embryos must have been introduced during the summer.

No such influence can be traced in the statistics given by Zenker (Müller, Dissert. Inaug. Erlangen, 1874).

As yet we know nothing with certainty about the age to which round-worms may attain, or, consequently, of the length of time they may be entertained by their host. It does not, however, seem probable that their term of life is very long, or that the time they remain in man is likely to exceed a year.

The cases, which are not unfrequently met with, where persons have for years expelled round-worms, are explained by constant fresh immigration of the animals taking place in a person who is in circumstances which are favorable to their introduction.

The number of worms in the intestine at the same time is very various. Usually there are several, sometimes a good many; more rarely these are present in numbers of one hundred or more; immense numbers are seldom met with among civilized people, and then they occur almost without exception in persons who are insane, and especially among the so-called "dirt-eaters." On the other hand, immense numbers of them seem to be the rule in tropical climates.

Cruveilhier[1] found the whole of the small intestine of a girl, who was an idiot, crammed with round-worms, and estimated their number at above one thousand. There are numerous accounts in our literature of similar cases, especially from tropical climates.

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[1] Dict. de méd. et chir. prat., Art. Entozoaires, p. 332 (Davaine).

Round-worms do not by their presence exclude other parasites from the intestines; on the contrary, it is not at all a rare thing to find several varieties together.

Consult the table of statistics on page 673.

Symptoms.

Round-worms, when present in small numbers in the small intestine, do not as a rule give rise to any phenomena. In large numbers, however, they may give rise to disturbances of different kinds.

Foremost among these phenomena we have itching of the nose, colic-like pains around the navel, boring and tearing pains in the abdomen, inflation of the region of the stomach, changeable appetite, and diarrhœa, with the expulsion of masses of mucus, which are occasionally tinged with blood. As external symptoms, we not unfrequently see swelling of the face, darkening of the eyelids, unequal dilatation of the pupils, foul breath, and general wasting. Nervous symptoms, such as irregular pulse, unpleasant dreams, grinding the teeth during sleep, and starting out of it in a fright, with pains in the limbs, are all said to be caused by the presence of the worm.

These symptoms are all very indefinite and but little characteristic; still when taken together, they are especially valuable as not belonging to any other disease.

To these may be added almost all those symptoms which in the chapter on the general pathology of intestinal parasites, and in that on tape-worms, have been put down as partaking of the character of reflex phenomena. Some of these are most dangerous —indeed, death is said in rare instances to have been produced in this way.

If, however, we impartially examine the accounts given of such cases, it will become evident that many diseases have been put down to the chance presence of these worms, with the production of which they had nothing to do. The most positive accounts of this sort come from the tropics, where, as we know, the round-worm is so universally common that it can be no cause for wonder when it is found to be present in all sorts of diseases. We may at the same time readily admit that a person, ill from any other cause, may suffer more on account of the worms, even though he had not previously been conscious of their presence. Especially in cases of disease of the intestines, among which in warm climates dysentery plays such an important part, may they readily cause an increase in the violence of the symptoms.

But if we refuse to allow that the round-worms play such an important part, it would be going too far to deny that their presence produces any symptoms at all, for there are cases known where the most violent symptoms ceased immediately on the expulsion of the round-worm.

Mosler[1] relates a specially interesting case of this kind, in which serious nervous attacks ocurring in a girl, who formerly had had an attack of convulsions brought on by fright, were almost entirely removed by the expulsion of a large number of round-worms. She was afterwards still subject to slight nervous phenomena of an hysterical character, so that the round-worms only acted as the immediate exciting cause of an outbreak of a disease already existing.

In the introduction we have already given a negative answer to the question, whether it be possible that intestinal obstruction could be caused by great numbers of live round-worms. We must not for a moment suppose that the large masses of entangled worms which are sometimes found at post-mortems, were formed during the patient's life. At the same time masses composed of dead worms mixed with other contents of the intestine, can very readily form an obstruction to any other body moving through the intestine.

It is still very doubtful whether, as has been supposed by some, round-worms can lead to incarceration of a hernia; they can hardly have a much different effect from that of any other half solid contents of the intestine.

Huber[2] has expressed the opinion that many of the symptoms put down to round-worms are caused by a peculiar irritating matter which they contain. This idea requires further corroboration before being ultimately received.

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[1] Virch. Archiv, 18, p. 242, 1860.

[2] Deutsch. Arch. f. klin. Med. VII. p. 450, 1870. Jahresb. d. Naturhist. Vereins in Augsburg, XIII.

Huber draws attention to a remark of Miram's,[1] that the examination of Ascaris megalocephala had twice caused him most unpleasant symptoms, such as sneezing, swelling of the puncta lachrymalis, hypersecretion of tears, with violent itching and swelling of the fingers. Huber himself, after examining twelve examples of Ascaris lumb., suffered from most troublesome itching of the hand and neck. Over the latter the skin was raised here and there in lumps, smaller lumps appearing on the forehead. His right ear swelled up, for an hour a plentiful secretion came from the meatus, and he had a most unpleasant sensation of pulsation in the right side of the head radiating from the ear. His conjunctiva became inflamed, accompanied with severe itching, the inflammation leading to chemosis in the right eye, and, finally, he had itching of the hands. After the lapse of an hour all these symptoms had quite disappeared. Huber considers that these symptoms were caused by the peculiar substance which gives off the strong smell, which is peculiar to round-worms, and which, as Leuckart supposes, is contained principally in the vacuoles between the muscular fibres.

In a large number of cases where round-worms were present, I have searched with the greatest care for any alteration in the mucous membrane of the intestine, without even once being certain of having found any; of course, small patches of ecchymosis are commonly met with without worms being present at all.

It is not much to be wondered at if the coils of intestine that contain the worms should sink by their weight to the lower part of the abdomen, and the vessels consequently be more filled merely as the result of post-mortem hypostasis.

Many of the cases where the mucous membrane is said to have been scraped off the intestine just above where the worms were found, may be explained by the mass being pushed forcibly before the scissors when the intestines were being laid open.

Round-worms often find their way into places between which and the intestine there is either normally direct or indirect communication, or, more rarely, into places to which they can only obtain access in consequence of some pathological change having taken place. Among the former, the stomach, œsophagus, nares, Eustachian tube, nasal duct, air-passages, the pancreatic duct, and the gall ducts, demand our attention; among the latter, the sac of the peritoneum, the pleura, the bladder, uterus, and vagina. Finally, they may be found in so-called "worm abscesses."

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[1] *Froriep*, Neue Notizen. VI. p. 108.

In by far the greater number of these cases the worm has found its way to its abnormal position after the death of the patient. During his life there have been no symptoms which would point to a long sojourn of the worm in the place where it is found. Neither are there any corresponding alterations to be found in the parts after death. Now neither of these two could be absent if the worm had commenced its rambles during the lifetime of the patient.

Occasionally, however, they do make their way into some of these organs; while into others it is of the rarest occurrence, even during the lifetime of their host.

Should round-worms make their way into the *stomach*, they as a rule cause violent sickness and vomiting, which latter generally causes their expulsion. They must of course not always be looked on as the cause of the vomiting; for, if the patient be attacked with vomiting from any other cause, they, like the gall or the contents of the small intestine, may also be accidentally brought up and expelled.

It is certainly extremely seldom that round-worms find their way into the *œsophagus* without previously having caused active symptoms of choking or vomiting.

This holds true of the *nares* also, through which they are sometimes expelled with other contents of the stomach when the patient is attacked with vomiting. We have, nevertheless, authentic accounts of their entry into both these organs, especially during sleep, without attracting the patient's notice. They cannot, however, give rise to any peculiar local changes, for they are soon taken notice of and expelled.

In rare cases they may from the pharynx make their way into the *Eustachian tube*, or from the nose creep into the *lachrymal duct;* but we have only isolated cases where this is authentically recorded as having happened during the life of the patient.

Bruneau (Davaine, p. 144) extracted a round-worm from the ear of a girl who had been attacked, while attending mass, with convulsions and pains in the ear, which was followed by the appearance of the worm in that organ. The symptoms ceased immediately when the worm was removed.

Of the two cases recorded where the worm entered the lachrymal duct, one, in which it is said to have occurred in a child three months old, is, to say the least of it, very doubtful. It is evident that in this case something else must have been mistaken for a worm. The second case, which occurred in an infant six months old, is also not free from doubt (Davaine, p. 144).

From the pharynx round-worms make their way into the *larynx* and *bronchi.* Of fourteen such cases collected by Davaine, in one only is it absolutely certain that this accident occurred during the life of the patient. In three of the other cases it is probable that the worm found at the post-mortem was the cause of the death from suffocation. Of the rest it is doubtful in some if the worm made its way into the air-passages during the patient's life ; in others it is quite certain that this did not happen till after death.

The eleventh case, given by Davaine, is the only one that can be looked upon as absolutely certain (Aronssohn, Archiv. génér. de méd. II. Sér. 1836. X. p. 44). A girl, eight years of age, was suddenly attacked, in the midst of health, with violent coughing, which soon increased to fits of actual suffocation. At the end of two hours, during which interval convulsions had come on, the little patient suddenly with great effort spit out a living worm, after which all the former symptoms ceased.

The symptoms caused by the entrance of a round-worm into the larynx resemble very much those produced by the entrance of any other foreign body of similar size. Unless the intruder is soon got rid of we will have violent cough, with breathlessness, palpitation, a feeling of dread, pain in the region of the larynx, attacks of suffocation, and even death.

Should a round-worm succeed in passing the larynx without causing great disturbance, the patient is not usually in immediate danger of death by suffocation, but he will suffer from cough and want of breath, either continuously or at intervals. Later on in the case, if the worm still be not expelled, the usual symptoms resulting from the presence of a foreign body in the larynx will set in.

For an account of these results I must refer the reader to the detailed account given by Riegel.[1]

Round-worms have in rare cases been found in the *pancreatic duct*, but there is no account given of any symptoms depending on it which occurred during life. It is probable that the worm had crept in there after the death of its host.

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[1] Vide Vol. IV., p. 501, et seq., of this Cyclopædia.

There is a specimen in the Pathological Museum in Kiel of a round-worm in the pancreatic duct.

Round-worms not unfrequently find their way into the *gall-ducts*, and have been found in the gall-bladder, and also in the biliary ducts, both external and internal to the parenchyma of the liver. Davaine enumerates thirty-seven such cases. In the majority of these cases the worms had crept in after the patient's death, as there were neither symptoms during life nor traces of their former presence after death. In the remainder there was either marked disturbance of the biliary system present during life, or more or less extensive anatomical lesions found at the post-mortem.

There can of course be no question but that the worms had entered the gall-ducts from the intestine, and had not been developed in them. Their entrance would of course be rendered easier by previous changes, such as the stretching of the gall-ducts by the passage of a stone. However, it is by no means improbable that young round-worms, which are remarkable for their great slenderness, can creep into the ductus (communis) choledochus.

Attention has already been frequently drawn to the love round-worms have for squeezing themselves through narrow openings.[1] Thus worms have been found encircled with rings of wire which had accidentally been swallowed, and, taking a hint from this, Stockbridge has constructed a "worm-trap."

The entry of a round-worm into the *gall-bladder* is followed, as a rule, with no symptoms or very slight ones. If it should become impacted in the cystic duct, then the usual symptoms of closure of this duct will set in.

If the worm finds its way into the *ductus choledochus*, the escape of the gall will be hindered; but if the duct is wide enough, it will not necessarily be completely stopped. If it quite fills up the ductus choledochus or hepaticus, then the same results follow as when they are closed from any other cause.

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[1] *Cobbold*, l. c., p. 312.

If the worm makes its way further into the biliary ducts, and thus enters the *parenchyma of the liver* itself, either a cyst-like dilatation is formed round it, which becomes filled with a catarrhal secretion, and communicates more or less freely with the biliary ducts, or it leads to a general dilatation of that section of the biliary ducts. The worm itself acts as a foreign body, either on the wall of the duct alone, in which case the mucous membrane becomes hyperæmic, spongy, and thickened, and gives out a copious catarrhal secretion, with occasional ulceration or perforation of the wall, or the inflammation spreads to the adjoining parenchymatous tissue, and an abscess is formed round the worm. This abscess may discharge its contents through the biliary ducts, or the inflammation may have the effect of completely cutting off its communication with them. It then occasionally happens that other secondary abscesses are formed, either in the neighborhood or at a greater or less distance away, which have no communication with the first.

By means of increased growth of the connective tissue around it, the abscess may become encapsuled, in which case the worm itself usually soon dies, and becomes disintegrated, and the contents of the abscess become thickened, and undergo cheesy or calcareous degeneration, with great diminution and contraction of the sac. Or the abscess grows larger, and opens into the surrounding parts. Thus cases have been observed where the abscess opened into the pleura, into the lungs, and through the walls of the chest, forming pyopneumothorax, and thus both the worm and the contents of the abscess have finally made their escape externally.

The various steps in this process give rise, as they occur, to more or less severe symptoms. Symptoms of hepatitis, such as fever, pain in the right hypochondrium, jaundice, diarrhœa—sometimes constantly, at others, returning at intervals—and convulsions, will be present, more or less marked.

It is not yet determined with certainty how long a round-worm can remain alive in the gall-ducts; it cannot, however, be very long. Worms, it is true, have often been found alive in the gall-bladder at post-mortems; usually, however, they soon die, become decomposed, and are then removed; or, as has once been observed,[1] they form the nucleus of a gall-stone.

It seems probable that such worm-abscesses may become wholly reabsorbed.

The observation of Virchow,[2] though usually supposed to be referable to the psorospermia, belongs, I think, without doubt, more properly here, both from the size and from the appearance of the forms he describes and pictures, and appears to me to be a most interesting example of this process having taken place. In the liver of an old woman, which was slightly atrophied, Virchow found a firm, roundish protuberance, having the appearance of sinew, and which measured from four to five lines in diameter. It was situated in a slight depression on the surface of the liver, which was surrounded by stunted liver lobules, which had a granular appearance. On making a section through the middle of the thick tendinous capsule, a cavity was opened. Its shape was almost globular, the diameter being from one-half to five-eighths of an inch, and its walls were wrinkled in places. The loose, cheesy-looking, dry contents consisted of a friable mass, in which a number of eggs were mixed up, sometimes occurring singly, at others in groups, some of which contained several dozen. These were covered with a tolerably thick, strong skin, which had a double outline, and was here and there slightly striated, outside of which there was a homogeneous, colorless, brightly shining, albuminous envelope, with a very undefined margin. Within these there was a fine inner skin. Some of them were quite filled with a roughly-granular yolk-mass, arranged in indistinct, roundish masses, through which could be seen a nucleolar body. Most of them, however, contained a light-gray, finely-granular substance, and only at one place, which was usually eccentric, was there a large round, brownish yolk-mass. Along with these there were others which were undergoing a sort of fatty degeneration. They were oval in shape, and measured, without the albuminous envelope, 0.002 Parisian inches, or 0.054 mm. The drawings, the description, and the measurements correspond most accurately to those of round-worm's eggs that have been kept a long time, and in some of which the process of segmentation has begun. In such eggs, also, we always find some that are undergoing fatty degeneration. According to Leuckart, the eggs of the round-worm measure from 0.05 to 0.06 mm. This mass was, therefore, what remained in the place where a female round-worm had perished; the ripe eggs that it contained, being placed in circumstances favorable to their being hatched, had begun to develop.

Round-worms can only get into the *bladder*, *uterus*, or *vagina*, when fistulous communications have been formed between these organs and some part of the intestine.

To these worms the power has been ascribed of occasionally piercing the intestinal wall, and thus gaining access to the peritoneum, and this, too, irrespective of whether the intestine was diseased or not. It was supposed that the worm could force itself between the fibres of the muscles, which closed again after it had passed. This idea was founded on the supposition that worms had been found within the peritoneum without any perforation of the intestine being present. An easier explanation is, that the perforation was not searched for carefully enough. We can neither allow to the worms the power of being able to *pierce the wall of the intestine*, nor to the intestinal wall the power of allowing it to pass through without leaving any trace of its passage behind it.

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[1] *Lobstein*, Catalogue du Musée anatomique de Strassbourg, No. 1987. (*Davaine*, p. 160.)

[2] Archiv, 18, p. 524, Plate X. Fig. 5.

It is quite another question whether or not the worms can *break through a diseased intestine;* and we do not think the question can be answered positively in the negative. In cases of tubercular ulceration we extremely often see the ulcer just on the point of perforating, or, indeed, having perforated the intestine, which has, however, become adherent to the neighboring organs at that point by partial peritonitis. The ulcers, too, in typhoid fever, not at all unfrequently open into the peritoneum. One of the principal exciting causes of such perforation is any solid body in the intestine, and round-worms which happen to be in the intestine may act in the same way as would a solid body. In general, however, at all events in the earlier stages of such a perforating ulcer, we see a true necrosis of the bottom of the ulcer precede the actual perforation, the necrosis being recognizable by the wall of the intestine at that point being stained with gall. We can therefore very well imagine that the round-worms may hasten the perforation of the intestine in cases both of tubercular and typhoid ulceration, when the ulcers themselves are nearly through, especially as we know by post-mortem experience what a small thing is sometimes sufficient to cause the thin layer that remains to give way. Their presence is probably specially hurtful as likely to precipitate such perforation before adhesion has taken place between the threatened portion of the intestine and the surrounding organs; and also, because they are likely, once they have escaped along with the contents of the intestine into the abdomen, to hinder by their active movements such adhesion, and thus cause a peritonitis that had been circumscribed to become general.

Perforation happens in a large percentage of all cases of ulceration of the intestines, without any worms being present, and not, as Leuckart supposes (II., 246), in very few.

In 250 cases of patients who had died of typhoid fever, Hoffman[1] found perforation in eight per cent.; and in Griesinger's 118 cases it occurred in almost twelve per cent.

The striking frequency with which round-worms have been found in cases of intestinal perforation in typhoid fever,[2] as well as the striking frequency with which the trichocephalus is met with in typhoid fever in general, can be explained by the very great prevalence of these parasites. The latter fact has, however, led to the trichocephalus being looked upon as the original cause of typhoid fever (Röderer u. Wagler, l. c.).

Just as in perforation of the intestine, so also in *round perforating ulcer of the stomach or duodenum*, the worm may make its way into the peritoneum, or, should the diaphragm be also perforated by the ulcer of the stomach, even into the sac of the pleura.

The case related by Müller[3] was probably one of this kind. A girl of sixteen years of age died after a very short feverish illness. At the post-mortem a hole was found in the posterior wall of the stomach about the size of a kreutzer, which corresponded to a similar hole in the diaphragm, which was loosely adherent to the fundus of the stomach. The serous membrane covering the posterior wall of the stomach, as well as that covering the diaphragm and base of the lung, was brightly reddened, and in some spots exhibited the appearances of hemorrhages. In the left pleura were a number of round-worms, of which two were still half in the stomach. To imagine, as Müller did, that the worms had pierced the perfectly sound structures, will be to give them credit for a truly wonderful performance. It further presupposes that the worms had all singled out exactly the same spot in the wall of the stomach for their attack. Davaine has already drawn attention to the absurdity of such an idea.

If round-worms are found in the abdomen, and there is no peritonitis present, they must have entered it after the death of the patient.

Worm-abscesses are not unfrequently met with. A worm-abscess is an abscess at any part of the body from which, along with its proper contents, worms are expelled. Such worm-abscesses may be formed in a variety of ways. The very large majority of those that have been observed (forty out of the forty-seven cases collected by Davaine) are situated, as Davaine pointed out, in one of two places, either at the navel or in the groin. Worm-abscesses at the navel occur almost always in children (of nineteen such abscesses, fifteen occurred in children), while those in the groin occurred almost exclusively in persons over fifteen years of age (of twenty-one such cases, nineteen occurred in persons over fifteen). Now these are exactly the places where herniæ are most commonly met with, umbilical hernia being the one chiefly met with in children, and femoral hernia in grown-up persons. A comparison of these facts inevitably proves that in the large majority of cases worm-abscesses are nothing more than herniæ, though it may be hard to say what was the exact part the worm had in causing the abscess. There are three distinct classes of such abscesses: first, those out of which round-worms alone, or mixed with pus, are expelled; second, those out of which round-worms and the intestinal contents come from the commencement; and, third, those from which the contents of the intestine alone are at first expelled, the worms not making their appearance till afterwards.

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[1] Untersuchungen über die pathologisch-anatomischen Veränderungen der Organe beim Abdominaltyphus. Leipzig, 1869.

[2] *Liebermeister*, Vol. I. pp. 150, et seq. of this Cyclopædia.

[3] Memorabilien. 1872, p. 448.

In the last of these classes the worms cannot possibly be the original cause of the abscess; in the two former they may have had some influence. From what has already been said, we may be allowed to doubt if they had any such influence, though possibly they may by their motion have hastened the giving way of the softened intestinal wall. It is also more than doubtful whether the occurrence of incarceration can be put down to their presence in the sac of a hernia, their action being probably quite similar to that of any other solid contents of the intestine.

Closely connected with the above-mentioned case of perforation consequent on pathological changes in the intestine, are such cases where the perforation takes place at a part of the intestine that is not covered by peritoneum, and a fistulous abscess is formed, which may make its appearance at the most different places. Some of the cases must be looked on as caused by the bursting of an already existing abscess into the intestine, such abscesses being by no means rare, even when no worms are present. To this category belong those cases of abscesses in the psoas muscle, in the kidney, and in the spleen, in which round-worms were found.

Diagnosis.

We are only in a position to diagnose the presence of Ascaris lumbricoides when we have either seen the worms expelled, or rather, since this only shows us that worms were there, and not that they are still present, if we can by means of the microscope demonstrate their eggs in the fæces. All other so-called characteristic symptoms are, especially if occurring singly, quite insufficient, and can at the most only point to the necessity of examining the fæces. It is also impossible to diagnose the presence of round-worms in any other organ of the body.

Treatment.

In spite of their size and comparatively great muscularity, round-worms do not offer any serious resistance to the efforts made to expel them.

The most favorite and never-failing remedy is *santonica*, the unexpanded flower-buds of different species of artemisia. It is given in powders, or, better, in a bolus or an electuary.

The medicine that is most in use at present is *santonine* or santonic acid, which, according to the researches of Rose, is the active constituent of the flower-buds, and which, except in very large doses, is quite innocuous. It is given three or four times a day, in doses of from one-third to one and a half grains, the latter dose only to a grown-up person, and should then be followed by a purgative. Castor-oil is not quite adapted for this purpose, for it has the power of dissolving the santonine, which is thus more readily absorbed; this, on account of the unpleasant symptoms it produces, is to be avoided. However, in spite of this, Küchenmeister recommends this combination.

Calomel has also been recommended, and latterly the Aristolochia bracteata by

Cobbold (l. c. 372), as well as the santonate of soda. According to Rose (Virch. Archiv, 16, p. 233), this last drug is very readily absorbed, and produces more violent general symptoms.

Prophylaxis.

In spite of the fact that these worms have now been well known for more than two thousand years, we are still unfortunately quite ignorant of the means by which they find an entry into their host. We are, consequently, not in a position to lay down positively the means by which they may, with certainty, be avoided. The only thing that will enable us to combat them properly is a thorough investigation of the conditions necessary for their development and for their introduction. We must, therefore, content ourselves with laying down some general rules for their prevention.

It follows directly, from what has already been said about their etiology, that cleanliness in the kitchen and in the house, with regard to our food and our drink, our clothes and our hands, here again stands foremost. Especial stress is to be laid on having a plentiful supply of pure water, and, if such cannot be procured, we must make use of no water that has not been previously either filtered or boiled, as in soup, coffee, tea, etc., etc.

A plentiful supply of pure water, which every one should by law be required to introduce into his premises, has become a vital requirement for the health of all large towns. For not only would this lead to a more general regard for cleanliness, and thereby to an increase of healthiness among the population, but would also serve as a barrier against the possible introduction of infectious matter, and also of the germs of the round-worm.

The town of Kiel, so large a percentage of whose population are afflicted with round-worms, has very bad fountains. Its water-works are fed out of ponds which can be approached on all sides, and which are situated in fields which are quite near to inhabited quarters of the town. If Davaine's view is the right one, the projected improvement in the water supply must have the effect of diminishing the percentage of persons who have these worms.

Ascaris Mystax.

This, which is the round-worm that infests cats and dogs, is much smaller than the one which infests man. The female attains to twelve ctm., the male to six ctm. in length. The worm is distinguished by two wing-like appendages composed of chitine, which extend from the head along each side, to a distance of from two to four mm., but are liable to great variations, as regards both their shape and their size. The eggs are somewhat smaller than those of Ascaris lumbricoides; at the earliest, in from three to four weeks, but usually somewhat later, the embryos become developed. The eggs have a great power of resisting external influences, and their development is not arrested in spirits of wine, chromic acid, or turpentine.

The method of transmission is, up to the present time, unknown. Leuckart found young round-worms in the stomachs of cats which resembled embryos, both in size and shape. Besides these, he found in the stomach and small intestine worms in every stage between this and the fully matured Ascaris mystax. He failed, however, to produce such forms by feeding animals on eggs containing embryos. (See also the account given in the section on Ascaris lumbricoides, p. 732.)

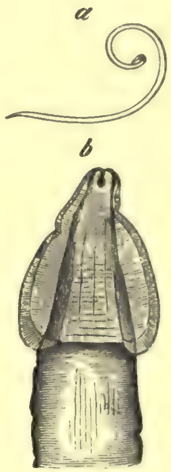

FIG. 44.

Ascaris mystax.
a, Natural size.
b, Head, showing the wing-like appendages (magnified).

For further particulars, I must refer the reader to Leuckart, II. p. 258.

The Ascaris mystax inhabits the small intestine of the cat, the dog, the fox, the lion, the lynx, and most probably some other animals. Up to the present time it has been found only five times in man.

Bellingham[1] was the first person who found this worm, and he described it under the name Ascaris alata, and at the same time drew attention to a case published by Pickells.[1] Cobbold[2] reported a third case, and at the same time pointed out the identity of the Ascaris alata and Ascaris mystax. He also vindicated Pickells' case from the attacks of Küchenmeister and Leuckart. A fourth case is related by Leuckart (l. c.). The Pathological Institution in Erlangen was presented by Dr. Böhm, of Gunzenhausen, with a female example of this worm, measuring fifty-five mm. in length, which was expelled by a young child.

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[1] Dublin Med. Press, 1839, No. 7.

From a clinical standpoint this round-worm is distinguished by its great activity, it frequently making its way into the stomach. One may often see dogs get rid of whole coils of them by vomiting.

Oxyuris Vermicularis.

(Thread Worm. Der Pfriemenschwantz.)

Natural History.

The thread-worm (Springwurm, Madenwurm) is a small, white round-worm, which is tapered off at both ends. The end of the worm at which the head is, is surrounded by a chitinous inflation of the skin, which makes it resemble in form the mouthpiece of a Turkish pipe. The opening for the mouth is closed by three nodular lips; it leads into the muscular œsophagus, which gradually grows larger posteriorly, like a club. Next to this comes a sharply defined circular portion, which is provided with a chitinous valve having three lappets — the so-called pharynx. The intestinal canal, which is the direct continuation of this, runs through the body almost in a straight line to the opening for the anus.

FIG. 45.

Oxyuris vermicularis. Natural Size.
1. Female.
2. Two males.

The *male* is from three to five mm. long, seldom more. Its tail comes rather suddenly to a blunt end, and is more or less bent or rolled up on the abdomen. The testicle, with the seminal duct, is a broad white tube, bent like a hook, and is situated in the posterior part of the animal; its efferent duct unites with the end of the intestine, and forms a short cloaca, out of which the spiculum, which is simple, slightly wavy, and bent like a hook at the end, often projects.

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[1] Transactions of the Col. of Phys. in Ireland, IV. and V.

[2] l. c., p. 322.

The bending and rolling up of the posterior end of the male worm is, as we may readily convince ourselves, always to be found in the worm during life, and would appear to assist it to embrace the female.

The *female* is from nine to twelve mm. long, and its posterior extremity is drawn out into a long awl-like tail, which is slightly wavy towards the end. The anus lies in front of the commencement of the tail; the vulva is situated a little anterior to the middle of the body, and leads into a short, very muscular vagina. To this is joined the uterus, the body of which is very short, and divides into two immense cornua, which separate, one running anteriorly and the other posteriorly. When these become full of eggs, they fill the whole interior of the animal so completely that all the other organs are almost entirely hidden.

The *eggs*, when looked at from above, are oval; from the side, however, they are seen to have one surface flat and the other arched, as well as one sharp and one rounded end. They usually contain a finely granular yolk. In the more mature females we find the yolk in all stages of segmentation, and sometimes the egg contains the so-called tadpole-like embryo (Fig. 47, *f* and *g*). This is an oval body, in the interior of which a light-colored streak may be seen—the commencement of the digestive apparatus, while at one end of it there is a tail, more or less long, according to the age of the

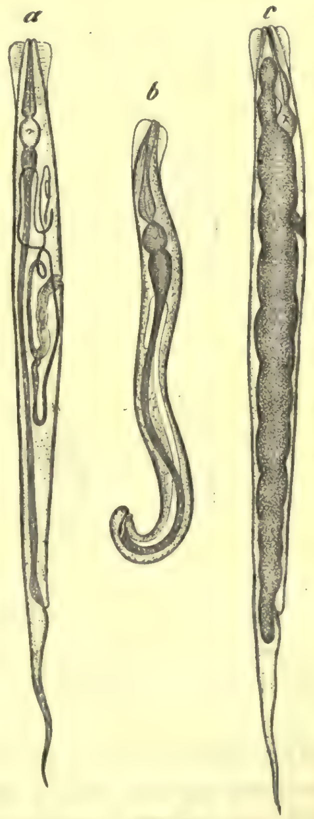

FIG 46.

Oxyuris vermicularis, magnified.
a, Mature but still unfructified female.
b, Male.
c, Female containing eggs.

embryo, which is folded up on the abdomen. At the ordinary temperature, but especially if placed on a warm microscopic slide, this tadpole embryo is sometimes observed to make rather lazy, at other times quick, jerky movements of rotation around its long axis. In the ripe eggs, found in the lower portion of the rectum and on the skin around the anus, we often see the embryo in a further stage of its development. It has then assumed the shape of a worm, is doubled up twice on itself, and its movements are much more active.

The Oxyuris vermicularis spends its whole existence, from the egg to maturity, in the intestinal tract of the same man.[1] Should a ripe egg be brought in any way into the stomach of a human being, by the action of the gastric juice, one place in the shell, situated on the arched side of the egg and towards its pointed end, becomes spongy or softened. If the embryo had already the tadpole shape, it will rapidly (in from one to six hours) ac-

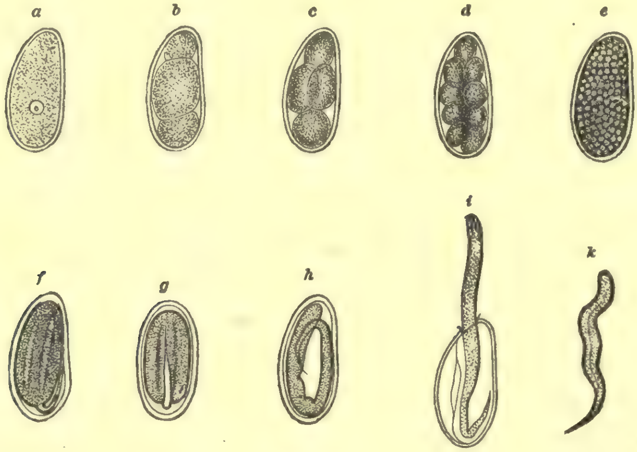

Fig. 47.

Development of Oxyuris Vermicularis.

a—e, Segmentation of the yolk. *f*, Egg containing tadpole-shaped embryo, seen from the side. *g*, Abdominal view of the same. *h*, Egg with worm-shaped embryo. *i*, Embryo escaping from the egg. *k*, Embryo that has escaped and is capable of motion.

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[1] The account given of the development is founded in a great measure on the investigations of Professor Zenker, in which I had very frequently the opportunity of taking a part.

quire the worm-shape, the anterior thick part of the body becoming thinner and more attenuated and elongating itself backwards. The tail grows thicker and longer, and becomes bent up a second time on itself as soon as it no longer finds space for its increasing length. The movements of the embryo now become more active; it presses its head against the softened place in the shell, breaks through it, and, little by little, at the expense of much effort, it finally struggles out.

All these occurrences may easily be observed under the microscope. The accompanying drawings were made from eggs which were corked up in a small glass bottle with a few drops of saliva and sugar and water, and kept for a night in the axilla. When placed on an artificially warmed microscopic slide the small worms crept further out of their shells; a portion of them had already escaped and were moving about with active serpentine movements.

The eggs of the Oxyuris require an amazingly short time for their development, provided they are placed under favorable external conditions. I once saw the metamorphosis, from the tadpole-shaped embryo to the worm-shaped embryo, completed in the course of about one hour. Under ordinary circumstances this usually takes from four to six hours; but it requires a much longer time if the circumstances be unfavorable. One of the most important of these conditions is the temperature, the development seemingly taking place quicker in proportion as the temperature of the medium in which the worms are placed more nearly approaches to that of the human body.

Whether eggs, in which the segmentation of the yolk has commenced, but in which the embryo has not yet been developed, can go on to their full development in the stomach, is very doubtful, but by no means impossible, for an embryo may become developed in them within a few hours.

In the mucus taken from the rectum, Vix[1] found eggs containing embryos, and near to them young worms escaping from their shells, and some also creeping about free. He therefore holds that no previous introduction of the eggs into the stomach is necessary, but that the young worms creep out of the eggs surrounded by the old worms, and go through all the steps of their development on the spot. Even if, owing to the trustworthiness of the observer, this occurrence must be considered as having undoubtedly taken place, still it must not be looked on as the rule, but rather as one of those quite exceptional cases which may happen under abnormal circumstances, as in chronic disease of the large intestine. Though I have often looked, I have never been fortunate enough to meet with a similar case.

As soon as it is free, the embryo, which has now become a very small, fine, delicate worm, would appear at once to betake itself to the upper portion of the small intestine, where it rapidly increases in size, obtaining its nourishment from the chyme and the intestinal mucus. As soon as it attains a certain size, the characteristic differences in the sexes begin to make their appearance. The male worm gets a short tail, which begins to taper quite suddenly; whereas in the female the tail becomes elongated into the shape of an awl. At the same time the development of the sexual organs commences inside the animal; almost the first thing that can be recognized in the male being the spiculum. The worm then casts its skin; this it accomplishes by alternately relaxing and then contracting itself; this is specially marked near the head of the animal, and by it the œsophagus gets several bends on itself; between the old skin and the new internal one, numerous bright, oil-like globules make their appearance, which seem to make the movements of the animal in its old skin much easier. At last the skin gives way, either in the middle or towards the anterior part, and the worm escapes.

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[1] Zeitschrift für Psychiatrie. XVII. Part 1. SA., p. 21.

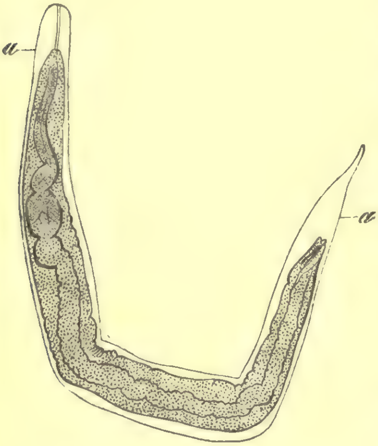

Fig. 48.
Male thread-worm casting its skin.

A young male worm, before casting its skin, measured 1.8 mm. long and 0.8 mm. thick. The œsophagus was 0.18 mm. long, the pharynx measured 0.06 mm., and the tail 0.1 mm. Another male measured 1.1 mm. long.

A young female, before casting its skin, was 1.97 mm. long, the vulva was situated at a distance of 0.8 mm. from the head, and the tail had a length of 0.4 mm. Other examples measured 1.78, 1.89, 1.91 mm. in length.

Such differences in size are chiefly due to the amount of contraction present.

This process of casting the skin, which has just been described, I myself have frequently seen (under the microscope) taking place in living worms taken out of the small intestine. It is quite easy to find animals which have not yet cast their skins.

It is not at all improbable that before the casting of the skin we have just described, the worms have cast it once, or perhaps even twice, previously; for we not unfrequently see worms, much smaller than these and in which there is still no indication of the sex, casting their skins.

Some of the young animals now spend a shorter or a longer time in the small intestine, and have sexual intercourse; others however, descend into the cæcum for this purpose. In favorable cases one finds large numbers of females in every stage of development, in the small intestine, in the cæcum, and in the vermiform appendix, in company with a like number of males. The latter seem to remain, as a rule, somewhat longer in the small intestine than do the females; at all events, we often find in the small intestines numerous males and only a few scattered females.

The young fructified females gradually collect together in the cæcum, and live there a considerable time, until they are fully grown and their uteri filled to bursting with eggs. They then commence slowly to descend the large intestine, and finally deposit the chief part of their eggs in the rectum; they occasionally even leave the latter and creep about on the moist skin around the anus.

There are numerous observations that make it probable that a quantity of eggs become periodically ripened and expelled. One of the chief is, that all the eggs present in a female are, in the same, or very nearly the same, stage of development. Further, the ovaries of those females which have expelled the greater part of their eggs have a healthy appearance and seem apparently to be still generating eggs. Finally, we find fresh quantities of sperm in the uterus and the vagina of such females, the presence of which must be put down to a fresh act of copulation.

The Oxyurides, both male and female, are finally expelled, purely mechanically, along with the fæces. Some of the females still contain eggs, others none; both males and females soon perish outside the intestine, if they have not already died within it.

The time requisite for their full development to maturity is very short. Leuckart[1] saw young ones, measuring from six to seven mm., expelled fourteen days after the eggs had been swallowed. In the processus vermiformis of a young child, five weeks old, I found numerous female worms, some of which already contained eggs. The time, therefore, that is necessary for their development, up to sexual maturity and the formation of eggs, cannot exceed five weeks, even if we suppose the eggs were introduced during, or immediately after, the birth of the child.

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[1] II. p. 336.

It cannot be denied that there is a possibility of the infection of the child with eggs from the mother during its birth. The conditions are by no means unfavorable for the occurrence of such an infection. It is of course impossible to determine, without having direct observations on the subject, whether this is a frequent mode of infection or not, and numerous difficulties stand in the way of making any such observation.

The Oxyuris vermicularis is only found in the intestine of man, from the jejunum to the anus; the young animals, in their various stages of development, and the mature males, inhabiting chiefly the small intestine; the pregnant and mature females chiefly the cæcum. This last place is their special habitat; for, when there are many worms present, it is here they are most plentifully found; and when there are but few, they are either only to be met with here, or they are scattered singly throughout the rest of the large intestine. The generally prevalent idea, and that which is upheld in all the books, that the Oxyuris inhabits the rectum, is entirely false.

Of course many differences will be observed, according as the age of the colony varies, and also according to whether the infection has taken place but once or a few times, or whether eggs are being constantly introduced. Certain pathological conditions, also, have a powerful influence on the number and the distribution of the animals; thus the greater portion of them are often simply mechanically washed out by an attack of diarrhœa. There are probably numerous other causes for such deviations. The description that has been given of what is the most usual condition is founded on a large number of post-mortem examinations.

The untenability of the view, that the Oxyuris has its seat in the rectum, should have been deduced from the fact that it is impossible to get rid of the worm by most thorough local treatment applied to the rectum; and it has been long known to the veterinary surgeon that the Oxyuris inhabits the cæcum of domesticated mammals.

Vix (l. c., p. 152), Gros, and Stricker[1] have long ago stated it to be a fact that the cæcum was the habitat of the Oxyuris. After them, Zenker[2] has repeatedly drawn attention to the same fact, and still, even at the present day, we find the false statement everywhere given.

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[1] Virchow's Archiv, 21, p. 360, 1861.

[2] Verhandlungen d. phys.-medic. Societät. Part 2, p. 20. Erlangen, 1870. Tageblatt der deutschen Naturforscherversam. zu Dresden, 1868, p. 140.

The number of Oxyurides that may be present at a time is very variable. Sometimes there are more, at other times less; their numbers are at times innumerable, and the whole mucous membrane of the intestine is so thickly covered with them that, as Vix has well expressed it, it looks just like fur. The total number of males and females is pretty equal; but in different places and at different times this equality may not be present.

The male worm long remained unknown. Even after Bremser had received examples of it from Sömmering, it remained quite a rarity. Zenker[1] was the first who demonstrated their frequency. Leuckart, however, from observations made on living persons alone, questions his statement that they are almost as frequent as the females. There are undoubtedly, as has already been said, many variations in their relative frequency. For, if only moderate or slight infection with the eggs has once taken place, the males are, after the lapse of a certain time, rarer than the females—indeed, there may be hardly any of them; for it appears that they die and are expelled sooner than the females. Indeed, the principal development of the female begins long after the male has fulfilled its duty. Usually, also, the males, which are much smaller, more delicate, and more transparent than the females, very easily escape notice. We can, however, without much trouble, readily prove that the numbers of one sex do not differ greatly from those of the other. The results of many cases where I have counted the animals, with this very end in view, prove this. In the small intestine I usually found numerous males and very few young females; whereas, in the cæcum, the females were numerous, with only here and there a single male; occasionally, however, they were met with in equal numbers in the cæcum. The processus vermiformis, as well as any intestinal diverticulum that may be present, is a favorite haunt of theirs. I once counted thirty-six male worms in the mucus obtained from one scrape of a knife along the surface of such a diverticulum. With my naked eyes I have found nineteen females (of whom fifteen were old and four young) and nineteen males in the processus vermiformis; at another time I found four old and five young females, and thirty males; and, on a third occasion, the numbers were seven old and twenty young females, and forty-six males. From their large size, it is hardly probable that a female was overlooked; but from their smallness, it is quite certain that many a male escaped observation.

The males are easiest found in the way already recommended by Zenker, viz., by firmly scraping off the mucus from a portion of the intestine, then spreading it out, with a three-quarter per cent. solution of common salt, on a plate of glass, and examining it either with the naked eye over a dark ground, or with a low magnifier and transmitted light.

The *geographical distribution* of the Oxyuris vermicularis is very great. For, corresponding to the extraordinarily favorable conditions for their distribution, their propagation, and their transmission (from one host to another), we find them in the far north as well as in the temperate and torrid zones. Hence we have accounts of the occurrence of Oxyurides from almost every place from which we have any medical accounts on the subject at all. As to the frequency of their occurrence in the different places, our general knowledge is still very insufficient. More searching investigations would, as may be seen from different accounts, of which we are even now in possession, show that these worms occur with almost incredible frequency.

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[1] *Küchenmeister*, l. c. p. 286.

In Dresden, Zenker found these worms in 2.1 per cent. of all the post-mortems he made, and in Erlangen in 12.13 per cent. In Kiel I found them in 23.2 per cent. of all post-mortems. (See the Introduction, p. 672.)

Etiology.

Oxyuris vermicularis can only occur in the intestine as a consequence of the entry into the stomach of ripe eggs, which have been furnished by some one who was himself suffering from the worm. There are numberless ways in which the first transmission may take place. In children it is usually brought about by the dirty hands of the mother or nurse ; we have also already pointed out the possibility of the infection taking place during birth (p. 758).

Once even a few animals have become developed in the digestive canal, there is no further occasion for the entrance of eggs from foreign sources ; for the conditions are most favorable for self-infection, ripe eggs of the host's own guests being carried to his mouth, and causing fresh infection.

Bakers, dealers in fruit, cooks, waiters, and persons having like occupations, have abundant opportunity, should they be afflicted with Oxyurides, of imparting them to others.

An emigration of Oxyurides from the rectum of one individual into that of another person, who is sleeping in the same bed with him, is nothing but a pure fable. The worms are incapable of motion on any dry surface, and perish quickly. Even the return of a worm which has strayed from the rectum on to the skin in the neighborhood, if it happens at all, certainly does so very rarely.

The number of Oxyurides present can be increased indefinitely by self-infection. With an increase in the number of the animals, the circumstances favoring self-infection also increase. Thus the numbers of mature females in the rectum are augmented, the consequent unbearable itching will become more and more frequent and severe, and will more and more often impel the patient to boring and rubbing with his fingers. Now, this itching is especially liable to occur at night, when the patient is warm and half asleep in bed, and in this condition the fingers or nails may very readily become covered with the eggs, or sometimes even with an entire worm. If, then, during the course of the night or morning the fingers should be brought to the mouth, or should the patient take any article of food in his hand, or should he indulge in the bad habit of biting the nails, which is by no means an uncommon trick with children, the eggs find a ready entrance into the mouth.

Professor Zenker has frequently demonstrated microscopically ripe eggs under the edges and at the roots of the nails, in persons who were afflicted with these worms, which fact I myself have often corroborated.

Hence it is that children, and grown-up persons whose habits of cleanliness are not much better, suffer so much from Oxyurides; and foremost among these, it would appear, are the inmates of lunatic asylums and persons who live crowded together into a small space, as in our orphan asylums. In like manner, those nations which are in a low state of civilization are, according to all accounts, afflicted with them to a very unusual extent.

It cannot be supposed that the eggs of the Oxyuris are conveyed into the stomach in water used for drinking, for the eggs soon perish in water. But it is just possible that, under peculiarly favorable conditions, they might be transmitted by the atmosphere, for they retain their vitality for some little time even when quite dry. But, if this occurs at all, it must be very rarely.

I have myself several times expelled a few of these worms, a short time after I had been engaged in any investigations on them, even though I took every possible precaution with regard to cleanliness; and Leuckart has noticed the same thing with regard to himself.

There can be no fear of infection from the use of green salad, for, as Vix showed, the eggs of the Oxyuris quickly perish in table-vinegar, even when mixed with an equal quantity of water.

There does not appear to be any such thing as a diathesis which predisposes a person to the acquisition of these worms. Chronic catarrh of the intestine, when accompanied with a plentiful secretion of mucus, would, however, appear to favor their thriving. On the other hand, diseases in which frequent watery stools are passed are evidently unfavorable to the Oxyuris, as indeed to all intestinal parasites.

Of one hundred and twenty-two persons over six months of age, who in 1873 were found free from parasites, thirty are put down as having some disease of the intestines; and of one hundred and sixteen who in 1874 were also found to be free, no less than fifty-one had some disease of the intestine, which in the large majority of cases was tubercular ulceration.

Pathology.

The Oxyuris is found in both *sexes* and at all *ages*, though women and children are somewhat more often afflicted than men.

The *occupation* of the individual does not seem to have any predisposing influence, nor does their frequency seem in any way to depend on the *season of the year.*

The *duration* of the disease is only limited by the death of the patient, and not unfrequently persons suffer from them their whole lives' time, even up to a green old age. On the other hand, the lifetime of an individual worm is undoubtedly a comparatively short one, and it is only by the continuous formation of new generations that the disease can be so protracted.

The youngest child in whom I found this worm was five weeks old; the oldest person in whom they were met was eighty-two years of age.

Symptoms.

The phenomena that are caused by the presence of Oxyurides must be divided into local and general.

Since the chief seat of the worm is in the *cæcum*, it is there that we would first expect to find evidences of their presence being injurious; but up to the present time nothing at all of the sort is known. We know just as little with certainty about the phenomena to which they may give rise by their presence in other parts of the intestinal tract.

We are not, however, able to say, whether if the worms were present in great numbers in the upper portion of the intestine, their very presence there might not give rise to some disturbance. Arguing from what happens in the first stage of infection with trichinæ, it seems probable that it might.

In the *rectum*, however, they always give rise to symptoms of such an annoying sort, that, among all the usual intestinal parasites, the Oxyurides must, in spite of their small size, be looked on as the very worst tormenters of man.

The presence of a single worm here and there gives rise to no disturbance, or the disturbance is only slight and occasional. In proportion as the number of the worms increase, these disturbances become more frequent and well marked; and if the number of worms present be very considerable, they become the source of constant inconvenience, which at times becomes quite unbearable. The worms descend into the rectum to lay their eggs, and cause, by their active boring movements, an unbearable tickling and a painful itching just within the sphincter and in the folds of the anus, which sometimes becomes so great as to make it almost impossible to keep quiet and bear it. It is at night, especially, that the inconvenience is most felt. This is not because there is any periodicity in the life of the worm, caused by the change from day to night, for "in their home darkness always reigns;" but it is then that external circumstances, such as the repose of the host, the warmth of the bed, and perhaps, also, the state of the digestion, incite the guests to greater activity; and, on the other hand, cause the host, whose attention is then no longer distracted by his daily work or occupation, to be more alive to the torture.

The patients endeavor, by every change in posture, by rubbing and boring with their fingers, and by walking about, to cause the troublesome sensations to cease. As a rule, however, they last for a considerable time, and only go away, sooner or later again to appear. Sometimes emptying the rectum brings ease, by removing mechanically the worms that were buried there. At others, however, it causes an increase of the inconvenience, for the mucous membrane, being deprived of the layer of mucus which served it as a covering, may become more intolerant of the attack of those animals that remain behind or which may afterwards descend from the upper part of the intestine.

The ordinary statement, that the sensations are produced by the worms creeping about the skin around the anus, is not corrrect. These worms are readily and quickly removed by the friction of the clothes or by the hands. The irritation and itching are caused by the creeping about of the worms inside the rectum, and it is on this account that such symptoms are so hard to be got rid of without special treatment.

In the female sex further inconvenience is caused by worms that have escaped from the rectum, creeping into the vulva and vagina, and there giving rise, from their serpentine and boring movements, to very great irritation of the sexual organs. This irritation may cause masturbation in young children, and is very likely to do so in older ones; indeed it has even been said occasionally to produce symptoms of nymphomania.

I once at a post-mortem found one of these worms in the cul-de-sac of the vagina. Dr. Westphalen has told me that he has, through the speculum, seen one in the os uteri.

Vix appears to think the worms do not themselves creep into these parts, but are carried thither by the hands.

Vallez[1] found one of these worms in the vagina of a patient suffering from pruritus vaginæ.

It is assuredly a very rare thing, if indeed it ever happens, for these parasites to make their way underneath the prepuce, or into the male urethra, for the conditions are too unfavorable for such an occurrence. But sympathetic irritation of the generative organs, consequent on the violent irritation of the rectum, can very readily occur, both in men and in women, without any actual invasion of the genital organs taking place by the animals themselves. As symptoms of such sympathetic irritation, erections, pruritus, and nymphomania have been observed with consequent manustupration and onanism. Davaine[2] has seen a tendency to venereal excesses, and Lallemand[1] involuntary seminal emissions, depend upon their presence.

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[1] Gaz. des hôpit. 1853. No. 113. (*Vix*, l. c., p. 6.)

[2] l. c., p. 213.

General constitutional disturbances are very frequently explained by both ancient and more modern writers as depending on the presence of these worms. We may, however, be allowed to doubt, provisionally, the soundness of this view. I need only, therefore, mention that they have been specially blamed for causing unnatural phenomena connected with the nervous system, such as loss of reason, hemeralopia, and many others. Any general disturbance of nutrition may be looked upon as consequent on the disturbances that have been mentioned as occurring in the sexual organs.

Diagnosis.

The diagnosis is very easy. The uneasy sensations about the rectum experienced by the patient should at once make us think of these worms, especially if they are most marked towards evening and when the patient gets warm in bed. We should also think of them if we observe children constantly putting their hands to the rectum or genitals. In general, too, we can readily see some of the animals on the surface of the fresh evacuations, on which, as long as they are warm, they move about actively with a serpentine motion. If very many worms are present, we can usually find one or two on the skin in the neighborhood of the anus. If we do not succeed in this, we can, as soon as violent itching and tickling occur, by means of a small clyster of about fifteen grammes of cold water, cause those that are in the lower part of the rectum to be expelled, and so make our diagnosis. Finally, the microscopic demonstration of their eggs is a very certain diagnostic mark. For this purpose we take some of the intestinal mucus, either from the end of the rectum by means of a spatula, or from any piece of paper that may have been made use of after a motion. It is, in general, easy to demonstrate the presence in these matters of a few eggs having the characteristic form, by spreading the mucus out on an object glass with a small quantity of a three-quarter per cent. solution of common salt, and then examining it.

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[1] Des pertes séminales involontaires. Paris, 1842, III. p. 116.

Many persons, who complain of having piles, have nothing but Oxyurides. These are the persons who become the apostles of the laxative method for curing piles, for by constant violent purging they are freed from the Oxyurides and at the same time from their supposititious piles.

Prognosis.

This is, of course, absolutely favorable, provided the treatment is carried out with a due regard to the actual habitat of the worms. As long as the rectum was looked upon as their chief seat, the treatment adopted was only followed by temporary benefit.

Pathological Anatomy.

The presence of these worms in the upper part of the intestine does not seem to cause any anatomical alteration in the parts. The chronic catarrh, that is so frequently met with in the large intestine, can hardly be looked on as caused by their presence, but, on the contrary, the plentiful secretion of mucus must be considered as a circumstance most favorable to their thriving in comfort.

In the rectum, on the other hand, and especially in its lower part, a state of irritation is produced by their active boring and serpentine movements, which is accompanied with redness of the mucous membrane and the production of ecchymoses. The greater part, however, of the changes that are found about the anus and in the rectum of such patients, are not to be looked on as directly due to the Oxyurides, but rather as the result of the violent and unmeasured efforts of the patient, by rubbing and boring, to obtain relief from the unbearable itching and tickling from which he suffers.

Lallemand (l. c.) thinks the ecchymoses are due to punctures produced by the ends of the tails of the worms; but they are much too delicate to be able to do this.

Treatment.

So long as the rectum was looked upon as the chief seat of these worms, from whence occasionally a few found their way to the upper part of the intestine, there could be no hope of thoroughly getting rid of them. Still, the years of constant suffering to which persons who are afflicted with these worms are doomed, are so unbearable, that a confession of being unable to relieve them must be looked upon as a sad proof of the poverty of practical medicine. A glance into the literature on the subject will show that even nowadays we may re-echo the complaints of Bremser.

He says (l. c., p. 168): "Just as these parasites are, on the one hand, to be counted among the most troublesome of all those that live at the expense of our bodies, so, on the other, do they at the same time belong to those which are the very hardest to exterminate. Their number is legion; and if, after we have slaughtered thousands, we lay our weapons aside for a moment, imagining ourselves safe from a fresh attack, new cohorts again advance with increased reinforcements. The fæces and intestinal mucus contained in the large intestine, behind which they hide themselves, serve them for breast-work and parapet. If one attacks them from the front with anthelmintics, these become so weakened by the long march through the small intestine that the worms only laugh at them. If we attack them in the rear with heavy artillery, the foreposts stationed in the rectum must certainly succumb; but the heaviest enema bombardment cannot reach those encamped in the cæcum: and so long as ever so few remain behind, in some hiding-place, they, from the amazing rapidity with which they are reproduced, soon again become a large army."

Since this worm inhabits the cæcum, all internal medicines which are absorbed in the small intestine are without effect. For this reason santonine seems to be of little use, too much of it being absorbed in the small intestine. They seem to possess an equal power of resisting the action of other anthelmintics, such, for instance, as koosso.

I have frequently found, in cases where patients were taking koosso and castor-oil as a cure for tape-worm, numbers of Oxyurides moving about most actively on the evacuation, evidently being driven out purely mechanically. An observation I made on a girl, who was under treatment for chancres and condylomata, speaks volumes for their powers of vitality. I found them creeping about quite lively under the dressing, which was made with a tolerably strong solution of carbolic acid.

The ordinary local treatment of the rectum, especially that with enemas, procures naturally only temporary benefit, for it is only able to destroy and remove the worms that happen to be in it at the time.

In order thoroughly to remove the worms, it is most judicious to try a combined treatment. Medicines are to be given internally, in order to reach the young animals which are still dallying in the small intestine, and either to kill them or to drive them onwards. Since we already know that by energetic purgation almost all the worms may be got rid of mechanically, so we ought to make choice of medicines that will effect this. Among these the neutral salts are most to be recommended, joined with copious draughts of water. We must, however, at the same time attack the large intestine; and, since clysters only serve to empty the rectum of its contents, we should make use of Hegar's new method for washing out the large intestine. By this method the whole of the intestine, as far as the ileocæcal valve, may be washed out. Simple water would do very well for this purpose—for in a short time it causes the worms to swell up and burst—but that it is not altogether without an injurious effect on the intestinal mucous membrane. Hence, Vix recommends a solution of castile soap in distilled water, or in rain-water, of the strength of from one to two and a half grains to the ounce. This has no unpleasant action on the intestinal mucous membrane, while at the same time it quickly destroys both the worms and their eggs. However, since the processus vermiformis is, as has already been mentioned, a very favorite hiding-place, especially for the young animals, and since it is hardly possible to empty it in this way, it might perhaps be necessary, in order to make the cure certain, to repeat the treatment after the lapse of some time.

By a series of most careful experiments, Vix has tested all the medicines usually used in enemata, and has found the above solution of castile soap to be the most effectual. For this purpose the ordinary soap of commerce varies too much in the amount of water it contains, and, furthermore, it is mixed up with a quantity of various free alkalies.

The method adopted by Hegar is a great step in advance of the ordinary treatment for diseases of the large intestine. The only apparatus that is necessary is an enema tube with an olive-shaped point, a piece of gutta-percha tubing about eighteen inches long, and a glass funnel. By placing the patient in various positions, on his hands and knees, on his belly, and on his back, the last especially with the thighs well flexed on the abdomen, we can fill the large intestine with fluid as far as the ileocæcal valve. The amount of pressure may be varied according as we hold the funnel high or low. Should hardened fæces be present, the fluid should be introduced under low pressure, and allowed to remain some time. About two quarts of lukewarm water are necessary for this purpose. Once the intestine is freed of its contents, we can wash it out with from three to four quarts of the soap solution. The solution is to be poured in slowly and under a low pressure; should any straining occur, we must stop for a short time till it ceases. In this way we can introduce from three to four quarts in from fifteen to twenty minutes.

It is obvious that we will only be able to persuade those persons to undergo so energetic a treatment who are really great martyrs to the worms, and wish to get rid of them at any price. One or two courses of purgatives will suffice for those cases in which but few worms are present and the inconvenience felt is slight and intermittent.

All treatment is, however, of only very transient effect, if we do not at the same time instruct the patient as to the ways in which the worms are introduced and their numbers increased. Any false modesty in this respect would *à priori* lessen the chance of a permanent cure. (Consult also the Prophylaxis.)

Prophylaxis.

From what has been said about the etiology of these worms, it follows that the first introduction, as well as their increase in numbers, always takes place by means of eggs that have been a comparatively short time laid.

Now it is hardly possible for any one entirely to prevent a few eggs being introduced into his stomach, but it is absolutely in every one's power to prevent their increase by self-infection. An anxious attention to cleanliness, with regard to the hands, and especially with regard to the nails, is the most certain protection. The oriental custom of washing the hands before every meal is deserving of more general adoption among us. The bad habit of munching, or even biting the nails, should not be tolerated. Children should, for many reasons, but especially as a protection against these worms, be watched, in order to see that they do not sleep with the hands underneath the bed-clothes.

It is also judicious to keep the members of the family and the whole household under observation, in order to see that if they have any worms they should get rid of them, as otherwise they are a constant source of infection for the whole neighborhood. All persons are to be suspected of having Oxyurides who, even half unconsciously, every now and then by their motions, or by rubbing themselves, show that they suffer from itching about the anus.

Trichocephalus Dispar.

Whip-worm. Peitschenwurm.

This worm was first described by Morgagni[1] at the end of the seventeenth century. But his account of it remained unnoticed; and it was rediscovered by Röderer[2] at the end of the eighteenth century. He gave it the name Trichuris, mistaking the head for the tail. Göze[3] was the first who recognized the real state of affairs, and gave it its present name.

The whip-worm, which is from four to six ctm. long, is composed of two very dissimilar portions. The pointed end of the animal is its head; the thin, thread-like anterior part of the body measures about two-thirds of its whole length; the true body is placed more posteriorly, and is shorter and much thicker, and comes to an abrupt end. On the abdominal surface of the worm and near its anterior extremity there commences a granular longitudinal band, which occupies about one-third of the circumference of the animal's body, and which is composed of short pieces of chitine set into the skin. This band reaches nearly to the true body of the worm; the short pieces of chitine then diminish in number and become more widely separated, and so become gradually lost.

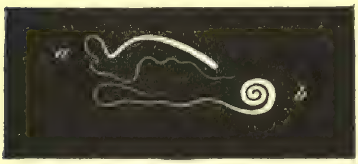

Fig. 49.
Trichocephalus dispar. Natural size.
b, Male. *a*, Female.

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[1] Epistolæ anatomicæ. XIV. Art. 42.

[2] De morbo mucoso. Göttingen, 1762.

[3] Versuch einer Naturgeschichte der Eingeweidewürmer, 1782, p. 112.

The intestinal canal consists of three portions, the œsophagus, the so-called chyle stomach, and the rectum. The œsophagus runs through the whole of the anterior portion of the body. Along almost its entire length runs a chain of very large darkly granular cells containing a dark nucleus, on one side of which the œsophagus is let in like a channel or gutter. To the œsophagus is joined the chyle stomach, which is a tolerably straight tube, which passes through the whole length of the posterior portion of the animal's body and ends in the short narrow rectum. This latter in the female opens externally near the end of the body; in the male it joins with the seminal duct and forms a cloaca. The posterior portion of the body of the female is only slightly bent, so that the shape of the animal reminds one of that of a hunting-whip. This portion of the body of the male is rolled up into a spiral form.

The *female genital opening* is situated just at the commencement of the thick part of the body, and leads into the vagina, which is very muscular, especially at first. This runs backwards in frequent convolutions, and opens into the uterus, which is larger than it in size, but has thinner walls. The latter organ is capable of very considerable development, and when quite filled with eggs occupies almost the entire posterior portion of the body. The tube which immediately joins on to it runs straight from the posterior to the anterior end of the thick part of the body, and then passes into the ovary.

The *male genital opening* unites with the end of the intestinal canal, and forms a cloaca; generally the spiculum, which measures about 2.5 mm., projects far out of this opening. It is surrounded by a broad, bell-like chitinous sheath, the inner surface of which is everted and thickly set with sharp-pointed teeth.

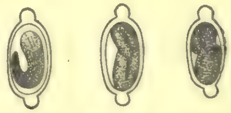

FIG. 50.

The eggs of Trichocephalus dispar in process of development.

The *eggs* are oval and of a slightly brownish color; they have at either pole a small, glistening knob, which gives them somewhat the shape of a lemon, and contains a finely granular yolk (Fig. 7). They require a long time for their development; at the end of several months at the earliest, and often not for a year and a half or more, we find a worm-shaped embryo developed in them. They have a great power of resisting the influence of their external surroundings. Thus they may become dried up or frozen without losing their vitality, though their development is thereby delayed.

Some eggs, that I had placed in water for their development, were, when the hospital in Erlangen was burnt down, in the year 1868, left in the open air; and the weather at the time being very cold, they remained firmly frozen for several days. This, however, did not prevent their subsequent development.

The ways and means by which we acquire the Trichocephalus dispar are still quite unknown. It is probable that it is introduced directly without the intervention of any intermediate host —the eggs that have been expelled, and in which, after the lapse of some months, an embryo has been developed, being brought, in some way or other, into the stomach, and the embryo then set free. It is probable that the embryos make their escape at the poles, for there it is that the most perishable part of the shell is situated, for we see in eggs that have lost their vitality the knob-shaped ends most frequently disappear, and the disintegrated contents of the egg welling out through the apertures which are thus left. Our experience of what happens in other parasites, as well as observations made on the Trichocephalus dispar in the small intestine, would lead us to think that the embryos escape while the eggs are in the stomach, and not, as Davaine supposed, that they do not escape until the eggs have reached the small intestine. In spite of the conditions for their entrance into the digestive system of man being apparently so very unfavorable, the continuance of the species is assured by its very great fruitfulness. Leuckart has estimated the total number of eggs contained in the uterus of a female worm at 58,000, and the yearly production from 300,000 to 400,000 at the very least.

From the experiments of Leuckart, it would appear that the embryos in the intestinal canal attain to their full sexual development in from four to five weeks.

Leuckart (l. c., II. p. 497) fed a lamb on eggs of the Trichocephalus affinis which contained embryos, and sixteen days afterwards found several hundreds of young slender worms, which measured from 0.8 to 1 mm. These worms were, to all appearances, young Trichocephali. A second case, in which he fed a pig on the eggs containing embryos of the Trichocephalus crenatus, corroborated the former one; for at the post-mortem, four weeks after the animal had eaten the eggs, there were found from fifty to eighty young Trichocephali, which had fully developed sexual organs, and measured from 10 to 30 mm. These experiments, though not amounting to absolute demonstration, support, so far as they go, the above-mentioned statement.

The Trichocephalus dispar lives in the *cæcum* of man. The number present is usually small; it is not, however, uncommon to find from seventy to one hundred of them; such immense numbers as one thousand, which Rudolphi found, are extremely rare. In cases where a large number are present, some of them are formed in the ascending colon, and even further from the cæcum. It is comparatively rare to find individual worms in the vermiform appendix or in the small intestine.

I have several times found a few Trichocephali in the small intestine; they usually appeared somewhat smaller and more delicate than those in the cæcum. Vix (l. c., p. 74) has also found one of these worms nine ctm. above the ileocæcal valve.

Wriesberg once found a Trichocephalus in the duodenum. The case where one of these worms was said to be found in the enlarged and degenerated tonsil of a soldier is more than doubtful.[1]

This worm is not peculiar to men, but is also found in monkeys;[2] moreover the Trichocephalus crenatus, found in pigs, is said to be identical with the worms found in man.[3]

The worm that Rose[4] found in the cæcum of a rabbit and took for a female Trichocephalus, was, without doubt, Trich. unguiculatus, for there is but little difference between the females of the several species.

This worm is found in both *sexes* and at every *age*, the youngest children alone excepted. The youngest person in whom I found the worm was two and a half years old; the oldest person was seventy-eight.

The *geographical distribution* of the Trich. dispar is very great, for it is found over the whole inhabited globe, and in some parts is extremely common. Thus it undoubtedly occurs in Germany, Italy, France, Denmark, England, and Ireland, and is often met with in North America.[1] It is extremely prevalent in Nubia,[2] Egypt, and Syria,[3] but very rare in the far north.

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[1] Microscop. Journ. 1842, p. 94.

[2] *Schneider*, Nematoden. 1866, p. 171.

[3] *Leuckart*, l. c., II. p. 468.

[4] Virchow's Archiv, 16, p. 235, 1859.

Cobbold says it is very rarely met with in England. This is quite contrary to the accounts given by Bellingham,[4] of Dublin, and Cooper,[5] of Greenwich.

Rudolphi[6] seldom failed to find it in the post-mortems he made in Berlin. Thibault had a similar experience in Naples. Zenker[7] found it in Dresden in 2.5 per cent., in Erlangen in 11.11 per cent., and in Kiel I met with it in 30.6 per cent. of all post-mortems.

Consult the Introduction, p. 672.

As to the *medical importance* of this worm, we cannot, on the whole, give a decided opinion. A few worms may, we think, be looked on as harmless guests ; if a large number are present, it is not at all impossible they may have an injurious effect. The way they are fastened to the intestine is of importance when we are considering this question. The thick parts of the body, as well as the posterior portion of the thin part, lie loose on the mucous membrane, or are surrounded by the contents of the intestine. The anterior portion of the worm is, on the contrary, firmly fastened to the mucous membrane, and it requires a pretty strong pull to get it away. The means by which the worm has such a firm hold, according to my observations, is that the anterior part of the body is disposed in several coils, in each of which it embraces a portion of the mucous membrane. It is probable that the longitudinal roughened band may be of considerable assistance to it in doing this, by acting as a sort of fastening apparatus. I could not in any case satisfy myself that the head had actually bored its way into the mucous membrane. I have, however, frequently observed it to be slightly embedded in the superficial layers, whereby a sort of furrow was produced. However, according to Leuckart[8] and Vix,[9] such a tunnelling under the mucous membrane would seem to take place. It cannot, however, be considered as the rule, and it must, therefore, be looked upon as questionable whether the mucous membrane is on this account more liable to the attacks of other diseases. I have several times found from sixty to one hundred Trichocephali in the cæcum, without there being any perceptible change in its mucous membrane.

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[1] *Leidy*, *Davaine*, l. c., p. 208.

[2] *Hartmann*, Naturgesch.-med. Skizze d. Nilländer, 1865.

[3] *Pruner*, l. c.

[4] Dublin Journ. 1838.

[5] *Davaine*, l. c., p. 208.

[6] Histor. natur. Entozoor. II. p. 91.

[7] *K. Müller*, Dissert. Inaugur. Erlangen, 1874.

[8] l. c., p. 465.

[9] Ueber Entozoen bei Geisteskranken, etc. p. 38, and Appendix (a reprint from the Zeitschrift f. Psychiatrie, XVII. Part I.), 1860.

Klebs also failed to find any penetration of the mucous membrane. The statement that the worms die very soon, on account of the cooling of the body after death, and are therefore no longer found with their heads sunk into the mucous membrane, is not the fact; for I placed a number of Trichocephali, which were removed from the cæcum forty-eight hours after death, in warm water at a temperature of 37° C. (98.6° Fahr.), and found them still living. I also made the same experiment with others taken from the vermiform appendage of the same patient, seventy-two hours after death, and they all displayed the most active movements.

Serious symptoms connected with the nervous system are said by different authors to depend, especially in children, on the presence of these worms. A doubt as to the correctness of this idea is certainly, up to the present time, quite allowable.

Roederer and Wagler were inclined to look upon typhoid fever as a morbus verminosus, from the frequency with which they found Trichocephali in the bodies of those who had died of this disease. This may be accounted for by the fact that it is only in patients who have died of typhoid fever that the intestines are always opened, and also because, the cæcum being thoroughly freed from its contents by the diarrhœa, the Trichocephali were more readily observed.

Since we know of no symptoms that depend on the presence of Trichocephali, we can only diagnose their presence by chance—that is, either by noticing a worm that has been expelled, or by finding the characteristic eggs when making a microscopical examination of the evacuations. The demonstration of the eggs is, as Vix[1] and Davaine[2] have already shown, very easy.

The object to be accomplished by the *treatment* is, of course, the removal of the worm from the cæcum. Up to the present time we have, however, no remedy that will do this with any degree of certainty. Bremser[1] once saw one of these worms expelled, along with round-worms and thread-worms, from a girl of sixteen years of age, who was under treatment for tape-worms. It is, however, probable that the worm was only carried away mechanically by the action of the purgative. If, in making a microscopic examination of the fæces, the quantity of eggs found is so great as to make it likely that a very large number of worms are present, we ought to try the effect of the Hegar-Simon's method[2] of washing out the intestine, combined with the use of anthelmintics.

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[1] l. c., p. 17, et seq.

[2] l. c., p. 209.

Santonine, when brought into contact with living Trichocephali, does not seem to have any effect upon them.

In the present utterly imperfect state of our knowledge as to the ways in which we acquire Trichocephali, we can, as far as concerns the prophylaxis, only repeat what has been said when speaking about the Ascaris lumbricoides, which this worm resembles in the long time it requires for its development.

Anchylostomum Duodenale (Dochmius s. Strongylus Duodenalis).

Dubini, Entozoografia umana. Milano. 1850, p. 102.—*Pruner*, Krankheiten des Orients. Erlangen. 1847, p. 244.—*Bilharz*, Zeitschr. f. wissenschaftl. Zoolog. 4, p. 53. 1853.—*Griesinger*, Archiv f. physiolog. Heilkunde. 13, p. 555. 1854; and Archiv der Heilkunde. 7, p. 381. 1866.—*Wucherer*, Deutsches Archiv f. klin. Med. 10, p. 379. 1872.—*Grénet*, Archiv. de méd. nav. VII. p. 209. 1867; VIII. p. 70.—*Kérangel*, ibid. X. p. 311. 1868.

This worm was first discovered in Milan by Dubini, in the year 1838, and shown to be a frequent parasite of the inhabitants of Northern Italy. It was afterwards found by Pruner and Bilharz in Egypt. Griesinger, on the strength of one post-mortem, declared it to be the cause of the disease called Egyptian chlorosis. Wucherer corroborated this statement by extensive examinations of the bodies of those who had died of the disease called tropical chlorosis.

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[1] l. c., p. 166.

[2] Vide p. 768 of this volume.

The Anchylostomum duodenale is a thickish, cylindrical worm. The end in which the head is situated is curved backwards; it has the shape of an obliquely-truncated cone, and is provided with a bell-shaped, chitinous-like capsule for the mouth. At the anterior margin of this are placed four strong, claw-like hooks, and two smaller ones are inserted into the opposite side. Still further inside the capsule two other pointed projections are fixed. The posterior part of the capsule passes into the very muscular pharynx. To this is joined the wide intestine, which is often filled with blood.

The *male* is from six to ten mm. long, and terminates in a three-lobed bursa, in which are placed two thin spicula. The male sexual apparatus consists of a long canal, which is arranged in numerous convolutions, which form the testicle and seminal duct; next to this is an oval or spindle-shaped seminal vesicle, to which is joined the very long and broad ductus ejaculatorius.

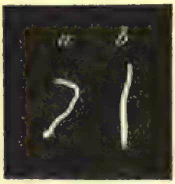

FIG. 51.

Anchylostomum duodenale; natural size. *a*, Male; *b*, Female. (Bilharz.)

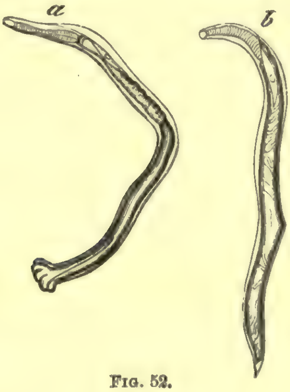

FIG. 52.

The same magnified.

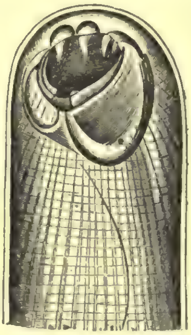

FIG. 53.

Anchyl. duod. Head showing the bell-shaped mouth. (Schneider.)

The *female* is from ten to eighteen mm. long, and sometimes as much as one mm. thick. Its posterior extremity has the shape of a pointed cone. The genital opening lies somewhat behind the middle point of the body, and through a short tube leads into the muscular, double vagina. To this is joined the very long, double uterus, with tubes and ovaries.

The *eggs*, which have a thin shell, measure 0.05 mm. long and

0.023 mm. broad; the process of segmentation has already commenced in them when they are laid. If they are placed in a damp place, in twenty-four hours worm-shaped embryos are developed in them, which make their escape, and, if placed in favorable circumstances, lead for some time an independent existence, and, by casting their skins once or twice, increase in size. Their further development is wholly unknown.

Wucherer (l. c.) has traced the development of the embryos to this point, when they always died.

Leuckart[1] concludes, from the experiments he has made with the Dochmius trigonocephalus of the dog, that the embryos of Anchylostomum duodenale develop into Rhabditis-like forms which do not resemble the parent, and are destined to lead an independent existence. These obtain their nourishment from various organized substances, and cast their skins once or twice. Once they have attained a certain size, it is necessary for them to be conveyed into the intestinal canal of man, where they grow larger, and, having cast their skin several times, finally attain the form of the Anchylostomum duodenale, and arrive at sexual maturity.

Leuckart has succeeded in almost absolutely proving that this is the history of the development of the Dochmius trigonocephalus.

It is, however, possible that there is still an intermediate stage in their development, for Bilharz observed in the submucous connective tissue of the intestine small spaces filled with blood, in some of which could be seen, curled up, a male, and in others a female Anchylostomum. These were alive, and had sucked themselves quite full of blood. Leuckart,[2] therefore, supposes that the Anchylostomum passes through its larva-like intermediate condition in these spaces, and afterwards, when the metamorphosis is complete, finds its way back into the intestine.

The ways and means by which this parasite is acquired by man cannot, of course, be laid down with certainty. Before this can be done, more careful investigations in those places where the worm is found are necessary. It is probable, however, that it is swallowed in the Rhabditis-form, along with dirty, slimy water.

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[1] Parasiten, II. p. 434.

[2] l. c. II. p. 444.

The worm inhabits the lower portion of the human duodenum and jejunum. Its anterior extremity is fastened between the transverse folds of the intestine, the mouth acting like a cupping-glass, and drawing a piece of the mucous membrane into its cavity. The body of the parasite lies in the direction of the chyle-stream, its back lying against the wall of the intestine. Several are usually met with in the same intestine, their numbers being sometimes immense. Bilharz found it almost in every dead body he examined in Egypt, the numbers present varying greatly, sometimes amounting to several hundred, at others to as many thousands. The number of males was, to the number of females, as 1 : 3.

In Europe the worm is only found in Italy, where Dubini found it in twenty out of a hundred dead bodies (in Milan). We know, also, that it occurs in the countries bordering the Nile, and on the Comoro Islands, which are situated at the northern extremity of Madagascar. In America it has been found, up to the present time, in Brazil and in Cayenne.

Léon Vaillant [1] declares that he has found it also in a gibbon. Now, since the two species of the gibbon only inhabit the Moluccas and Java, so Anchylostomum must also be endemic there. (See what was previously said on the geographical distribution of geophagia.)

The statement that this worm is met with in Iceland is untrue. (Leuckart, l. c. p. 411.)

It is probable, even, from the very great distances that separate the places where this parasite has been found, that its distribution is very much larger than what has been given. But since the idea that was first put forward by Griesinger, viz., that the Anchylostomum duodenale is the cause of a widely spread disease, has been almost raised beyond doubt by Wucherer, we must expect that these parasites will be found wherever this disease prevails. More extensive investigations in this direction are greatly to be desired.

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[1] Nouv. dictionnaire de méd. etc. red. Dr. Jaccoud. XIII. p. 335, 1870.

Indeed, there can hardly be a doubt raised against this idea, since Wucherer, and after him many other members of the profession, have demonstrated its soundness by numerous post-mortems.

Marchand (Thèse, Montpellier, 1869)[1] alone, of modern authors, questions the view held by Griesinger, declaring that in Cayenne he has not always found the parasite in cases of this disease. It is obvious, however, that he includes under the name "tropical chlorosis" other anæmic conditions. For at the post-mortem he frequently found in the "liver and spleen, the well-known signs of malarial disease," whereas other observers are almost unanimous that in tropical chlorosis the liver and spleen are seldom enlarged—on the contrary, are generally small and atrophied. It would further appear that most of Marchand's observations were made on transported Europeans, who, out there, are the more liable to be attacked with malaria. Finally, it is on this very same ground that Kérangel has made his corroborative investigations.

The accounts we have of the disease, which we must look upon as depending on the presence of the Anchylostomum duodenale, reach back into the seventeenth century.[2] The disease itself has been described under the most various names: Tropical chlorosis, Egyptian chlorosis, geophagia, dirt-eating, allotriophagia, mal d'estomac, cachexie aqueuse, cachexia Africana, cachexia Americana, hypoæmia intertropicalis, oppilaçaõ; and many other names have been made use of.

The *geographical distribution* of the disease is not yet quite determined, as many authors have confused it with other anæmic conditions, notably with malarial cachexia. We at all events know that it is confined to warm climates, and as yet has not been observed further north than Italy.[3] It is widespread in the West Indies, Guiana, Brazil, and the Southern States of the Union (Louisiana, Alabama, Georgia, Florida, and South Carolina); in Algiers, in the Comoro Islands, and in Senegambia.[4] The only accounts we have from Asia are from the Indian Archipelago.[5] In Europe it has up to the present time been seen in no place, except in Italy.

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[1] Jahresbericht, 1869, I. 313.

[2] Wucherer, l. c.

[3] Compare *Hirsch*, Histo.- geograph. Pathologie, I. p. 558, 1860, where the reader will also find a collection of the literature on the subject.

[4] *Thaly*, Archiv. de méd. nav. 1867. (Jahresbericht, 1867, I. p. 391.)

[5] *Van Leent*, Geneesk. Tijdschr. voor de Zeemagt. (Archiv. de méd. nav. 1867. Jahresbericht, 1867, p 385.)

As far as *race* is concerned, all accounts agree that it is principally the colored races that suffer from the disease. Next to these come the numerous crosses that occur between the white and colored races in the Brazils and the West Indies. In Cayenne, however, Kérangel saw Coolies, Chinese, Arabs, and Europeans also attacked. In the Indian Archipelago, the Malays, in Egypt both the negroes and the native Egyptians, are victims of the disease. It is only in the north of Italy that the disease has ever made any great inroads on a white nation.

From these facts it seems probable that it is not so much the peculiarities of race that predispose to the disease, but rather unfavorable external circumstances, especially with regard to food and drink.

Thus all the nations that have been mentioned live in their own lands under conditions that are very favorable for the introduction of Anchylostomum duodenale into the digestive canal. The worm is only met with in hot climates; in all probability it is developed in damp, slimy places, where for some time it leads an independent existence in a Rhabditis-form before it reaches a habitation, viz., the human intestine, which is suitable for its further development. From the dearth of good drinking-water, the poor people, who, as a rule, stand very low in the scale of civilization, are compelled by the heat and by thirst to drink a dirty, muddy water, which is drawn out of puddles and cisterns, whereas the more favorably circumstanced portion of the population can guard themselves from infection by purifying the water, or can avoid such sources of supply altogether by making extensive use of coffee, tea, and foreign imported fermented drinks.

Pathology.

Symptoms.

The chief characteristic symptom of the disease, which is caused by the Anchylostomum duodenale, is a greater or less degree of anæmia.

The disease begins so insidiously that we are unable to fix any time for its commencement.

In less severe cases the chief symptoms are paleness of the general integument and of the mucous membranes, fatigue after slight exertion, occasional dyspepsia, a tendency to palpitation of the heart, with quickened pulse, and murmurs in the jugular veins. The general nourishment of the system is often not interfered with, and the quantity of fat in the body may be considerable.

Even in more severe cases, the general nourishment of the body may at first be but little interfered with, the patient not becoming emaciated till later on in the disease. In these cases the first symptoms are, constantly increasing debility, a dislike to all exertion, and excessive sleepiness. Next comes paleness of the skin and mucous membranes. In negroes, the color of the skin becomes paler, more gray, and at the same time shrivelled and dry, and it peels off. The extremities are cool, and the patient is constantly complaining of feeling cold.

In rare cases slight feverish symptoms may be present.

After these general derangements of the system, the next most striking symptoms are those connected with the *digestive organs.*

In many cases dyspeptic symptoms appear rarely; loss of appetite alternates with truly ravenous hunger. Accompanying this, there is usually, if not always, a great desire to eat all sorts of things, which are usually most indigestible, such as mortar, earthen-ware, wood, coal, wool, cloth, and many others. The evacuations are often colorless. Violent pains in the stomach, or at all events more or less feeling of weight in the epigastrium, are usually present.

The characteristic effects of anæmia on the *circulation* are usually present at an early stage of the disease. While at the commencement the pulse remained slow and soft so long as the patient remained quiet, slight exertion is now sufficient to make the pulse rapidly mount up and to produce violent palpitation. As the disease progresses, the pulse becomes permanently quick; the palpitation, accompanied with unusually violent action of the heart, is constantly present, the second sound becoming audible at some distance from the patient. A systolic murmur is heard when the stethoscope is placed over the heart or the larger arteries, and loud rushing and buzzing sounds in the veins of the neck, giddiness, singing in the ears, black spots before the eyes, and headache, are constantly present.

The patient sooner or later begins to complain of shortness of breath, which is increased to the most violent dyspnœa on the least exertion.

The urine is usually secreted in abundance; its color is very pale, and it does not usually contain any albumen.

In female patients menstruation ceases, conception seldom occurs, and, when it does, the child is brought into the world in a pitiable condition.

The *duration* of the disease varies greatly. Some cases run a tolerably acute course, and end fatally in a few weeks; but as a rule the disease lasts for some months, and, if the patient gets plenty of nourishing food and saves himself in every way, it may even continue for years. If, on the other hand, the patient exerts himself much and gets insufficient or bad food, his strength is soon gone, and other diseases, even though not serious ones, by which he may be attacked, run a severe course, or imminently threaten life.

The patients now and then recover, especially by a change of climate and of all their external surroundings. As a rule, however, in severe cases the symptoms gradually increase in severity, the patient becomes dropsied all over, is attacked with profuse diarrhœa and uncontrollable vomiting, and finally dies.

Thus the intensity, the duration, and the result of the disease depend chiefly on the *amount of blood lost*, and therefore on the number of the worms that are present in the intestine. The quantity of blood lost is not confined to what the worms abstract directly from the body for their sustenance. For it would appear that they now and then change their position, and that the wound which they leave behind them, which closely resembles the bite of a leech, is the source of constant slight hemorrhage.

Pathological Anatomy.

The body is usually emaciated, though now and then it still retains a considerable quantity of fat. The subcutaneous cellular tissue is usually œdematous and the muscles pale and flabby. The internal organs are all very white and bloodless. Œdema of the meninges of the brain, hydrothorax, hydropericardium, and ascites, are very frequently present.

The heart is either of normal size, or is found somewhat enlarged, especially on the left side; its substance is pale, flabby, and easily broken down (anæmic fatty heart). Its cavities contain a very watery blood, or moist, flabby clots of blood or fibrin.

The liver and spleen are as a rule smaller in all their diameters than usual, sometimes to a very considerable extent. Their substance is pale, bloodless, tough, and flabby, but rarely in a state of amyloid degeneration. It is only when the disease has been complicated with, or when the patient has formerly suffered from, malaria, that they are found enlarged and displaying the characteristic changes of that disease. The kidneys are usually pale and shrivelled up, seldom amyloidly degenerated. The pancreas was found enlarged by the Brazilian doctors. The stomach and wall of the intestine are very pale; in the duodenum and jejunum is found a bloody mucus, and sometimes even fresh blood, which is partly coagulated; the mucous membrane is thickened and covered with numerous ecchymoses, some of which are as large as a lentil. A worm is found attached to many of these places where the intestine is filled with blood. Bilharz has occasionally noticed flattish elevations of the size of a lentil; these were of a livid reddish-brown color, and corresponded to spaces situated in the submucous tissue, which were full of blood, and in each of which was a curled-up Anchylostomum duodenale. In many places a white spot, about the size of a pin's head, could be seen in the middle of these elevations, in the centre of which was a hole having the diameter of a needle, which penetrated into the submucous tissue. (There are perhaps places where the worms situated in the submucous tissue have made their way into the intestine, and not, as Bilharz supposes, where the worm had made its way from the intestine into the submucous tissue.)

Diagnosis.

The diagnosis of this disease should properly be founded on its etiology. However, no one has up to the present time succeeded in finding either the worm itself or its eggs in the evacuations. If, however, the number of the worms present in the intestine be at all considerable, it must undoubtedly be possible to find the eggs under the microscope. The only other eggs with which they might be confounded are those of the Oxyuris vermicularis. The latter, however, may be distinguished by their peculiar form (v. p. 753), and also because when expelled they contain a tadpole-shaped embryo.

Wucherer appears to be the only person who has tried, and that unsuccessfully, to find either the Anchylostomum or its eggs in the evacuations. The search is rendered difficult, because the eggs being laid high up in the intestinal canal are there added to its contents, and are therefore thoroughly mixed up with the fæces.

However, the diagnosis may be made, without demonstrating the presence of the eggs, from the above short account of the symptoms, by excluding all other diseases which are known occasionally to give rise to a similar cachectic condition. Under this head we would notice ordinary chlorosis, leukæmia, the cachexia of cancer, progressive pernicious anæmia, and possibly that rare form of Addison's disease in which there is no bronzing of the skin.

The first of these does not need any further notice, as there is no difficulty in the differential diagnosis. The last occurs too rarely to require it to be taken into consideration at all. The disease, known under the name of "progressive pernicious anæmia," as it has lately been described by Biermer, Gusserow, Immermann, and Zenker,[1] certainly possesses such an extraordinary resemblance that we are likely at once to think of the disease produced by the Anchylostomum. But Immermann especially mentions that this worm was not found in the intestine. Further, in that enigmatical disease there is a great proneness to capillary hemorrhages, which are not among the symptoms that have been given of the tropical chlorosis.

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[1] Deutsches Archiv f. klin. Med. 13, p. 209, 1874. *Immermann*, in a later volume of this Cyclopædia.

The resemblance between progressive pernicious anæmia and the disease produced by the Anchylostomum is most exceptionally striking. I was unfortunately unable to get access to Biermer's communication on the subject (Schweizer Correspondenzblatt, II. 1, 1872). The total number of cases that have been observed up to the present time is twenty-three, of which one occurred in Dresden, and all the others in Switzerland. We at once involuntarily think of the proximity of Northern Italy, where the Anchylostomum was discovered.

The diagnosis becomes more difficult when complications are present. Even in such cases the marked anæmia, with its accompanying phenomena, not being a characteristic symptom of the complicating disease, will serve to put us on the right track.

Above all, we must take into consideration the local characteristics of the place. In parts of the world where tropical chlorosis is endemic, we must direct our special attention to the peculiar symptoms of this disease, even though at the time some other disease may be the more pressing ; for experience has taught, that when this condition of hypoæmia is present, if the patient contract even a mild disease, it is very likely to run a severe course.

Prognosis.

The prognosis depends on the condition in which the patient is when he first comes under treatment. If well-marked prostration be not already present, and if the true cause of the disease be not overlooked, it should be favorable. For we may hope, once we have got rid of the parasite, by suitable strengthening treatment, to restore the patient. If any complications are present, the prognosis is much more unfavorable, for they, as a rule, increase the debility, and so hasten on the fatal end.

Treatment.

We must look on the removal of the cause of the disease as being the first indication for treatment, for except we remove this blood-sucking parasite all our other labor is in vain. Anthelmintic remedies are therefore among the first that we should try. As, however, the Anchylostomum duodenale does not live on the contents of the intestine, but on blood taken from the blood-vessels, we need only consider such medicines as are either able to kill the worm by coming into contact with its epidermis, or which, when taken up into the blood, act as a poison to it without injuring the patient's health. We have, however, even at the present day, no medicine which is known to have this power, and we are therefore compelled to fall back on such remedies as are recommended by theory or have been suggested by experience. Among the latter we must mention those medicines which have been greatly praised for their effect by the Brazilian doctors, such as the milky juice of the Gammeleira (ficus doliaria), and a preparation called "doliarina," which is procured from it; then the milky juice of the Carica dodekaphylla, Velloso (Jaracatia, Martius). Griesinger recommends the use of calomel and oil of turpentine.

Benzine and picric acid both deserve a trial; the former both on account of its energetic power of killing parasites, and because the parasite in the present case is situated so high up in the intestinal canal; the latter because it can be taken up into the blood, and thus carried to the parasite.

Neither while we are administering anthelmintics, nor afterwards, should we neglect to put our patient under strengthening treatment, with the view of keeping up the general health; for this object, iron and quinine, with plain, generous diet, are strongly indicated.

Prophylaxis.

The problem of prophylaxis is a double one. First, as concerns the individual: he must, when in any place where Anchylostomum prevails, observe the greatest care and cleanliness with regard to every article of food and drink in general; but he is to be especially warned against using any water that has been procured from puddles or cisterns, as it is more than probable that it is in such water the young animals are introduced into the body. If no other drinking water can be procured except from cisterns and suspected fountains, this must never be made use of until after it has been most carefully filtered.

The second problem is one for the sanitary authorities. In countries where such immense loss of power and life is caused by the Anchylostomum, as in Egypt, where, according to Griesinger, the fourth part of the whole population is more or less afflicted, its object should be to procure a plentiful supply of pure drinking water, either by laying down a system of water-pipes or by digging wells. The cost of such measures, which is always considerable, would be amply repaid by the increased health of the inhabitants, and their consequent increased power to work and earn wages.

Echinorrhynchus Gigas.

(Riesenkratzer.)

This is a white, cylindrical worm, tapering somewhat towards its posterior extremity. It has a short thin neck, and has the power of retracting its head, which is set with six rows of powerful hooks. The male measures as much as ten ctm., the female as much as thirty-two ctm. in length.

This worm is frequently found in the small intestine of both the wild and the domesticated pig. Its head is sunk deeply into the wall of the intestine, to which it is very firmly attached. If it is forcibly torn away, there remains a tolerably deep, round hole, the margins of which are thickened and infiltrated. Corresponding to this, on the outside of the intestine, there often projects a flat roundish swelling.

It has only once been found in man, by Lambl,[1] in Prague. This worm, which was still quite young, was found in the small intestine. It measured 5.6 mm. long, 0.6 mm broad. The head was 0.36 mm. long, and the hooks were arranged in eight rows.

Welch[2] found an Echinorrhynchus encapsuled underneath the mucous membrane of the jejunum of a soldier who had served fourteen years in India.

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[1] Prager Vierteljahrschrift. 61, p. 45. 1859.

[2] Lancet. 1872, p. 703.

LARYNGITIS PHLEGMONOSA,
PERICHONDRITIS LARYNGEA,
ULCERATIONS AND TUMORS,
AND
NEUROSES OF THE LARYNX.

VON ZIEMSSEN.

LARYNGITIS PHLEGMONOSA.

Œdema Laryngis. Abscessus Laryngis.

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I adopt the designation, laryngitis phlegmonosa, proposed by Bouillaud, for those inflammations in the larynx which run their course principally in the submucous connective tissue, without, however, being exclusively limited thereto. To simplify matters, while considering them, I shall at the same time take up those forms of laryngeal œdema which are not of inflammatory origin.

The history of phlegmonous laryngitis and its sequelæ dates back to the beginning of this century. It is true that Morgagni describes the œdema attending inflammation and ulceration of the laryngeal mucous membrane; but the first real nosography of œdema is that given by Bayle in the memoir above referred to, which was presented to the Medical Society of Paris in 1808. Bayle's œdema glottidis is a serous infiltration of the submucous connective tissue, non-inflammatory in its origin. The establishment of the fact that inflammatory œdema of the laryngeal entrance is much more frequent than that which is non-inflammatory, was reserved for later investigators, as well as the introduction of the more appropriate term, œdema of the larynx, instead of the less appropriate one, œdema of the glottis.

Besides the term, phlegmonous laryngitis, which, as above stated, was proposed by Bouillaud, and which was intended to indicate the identity of this process with the phlegmonous inflammations of other mucous tracts, especially those of the pharynx, inflammation of the laryngeal submucous tissue has also received other names, which are intended to indicate the nature of the inflammation and the origin of the œdema. Such are: angine laryngée œdémateuse (Bayle), laryngitis submucosa purulenta, and sero-purulenta (Cruveilhier), laryngite purulente (Miller), laryngite œdémateuse (Mandl).

Under the designation, *œdème sousglottique*, sub-glottic œdema of the larynx, Sestier, Cruveilhier, and, of late, Gibb, refer to an œdema of an acute inflammatory character, limited to the region below the cords, which threatens life by rapid closure of the lower laryngeal cavity.

Etiology.

Phlegmonous inflammation of the larynx is almost always a secondary affection. It may be caused by the most various primary affections of the larynx and its surroundings. Perhaps the most frequent amongst them is the extension of purulent inflammations of the laryngeal perichondrium, or of the soft parts of the mouth and pharynx, as well as the irritating influence of foreign bodies.

The submucous connective tissue not unfrequently participates, to a serious degree, in *the inflammations of the mucous membrane.* Primary catarrhal laryngitis may, on the intervention of some new injury, especially in case of taking cold anew or over-exerting the voice, extend in depth beyond the mucous membrane, and lead to serous exudation into the loose cellular tissue of the ary-epiglottic folds, etc. This termination of acute catarrhal laryngitis has already been alluded to under the head of Catarrhal Inflammations. (See volume IV.)

The submucous inflammation is found more frequently following *diphtheritic* laryngo-pharyngitis, both the primary epidemic form, and the secondary diphtheria, complicating other, especially infectious diseases.

The pharyngo-laryngitis caused by severe *chemical* or *thermal irritants*, which always runs a highly acute course, is likewise seldom confined to the mucous membrane, but usually also implicates the submucous tissue in the inflammatory changes produced, whether these changes result in the formation of crusts on the mucous membrane of the pharynx and mouth of the larynx, or not. The most common injuries of this kind result either from *cauterization* with caustic alkalies or concentrated mineral acids, or from *scalding* with hot fluids (more rarely by the inhalation of a flame, or of hot air, in cases where the clothes are on fire or in a burning house).

Scalding of the mucous membrane of the fauces and laryngeal entrance is most common in England. The bad practice prevalent among the poorer classes, of allowing children to drink out of the tea-kettle, when no other utensil is at hand, results in the children's sometimes attempting to drink out of a kettle filled with *hot* water, and thus scalding themselves more or less severely according to the temperature of the water. Such cases were reported as long ago as Ryland's time.

Cauterizations most commonly result from the introduction of sulphuric or nitric acid, or of soda or potassa lye, or caustic ammonia (Ruehle), etc., into the throat, either with suicidal intent or by accident.

Furthermore, *mechanical irritation* of the mucous membrane by *foreign bodies*, especially pointed bodies, such as splinters of bone or fish-bones, which become entangled in or puncture the membrane about the mouth of the larynx, and are not soon removed, results in an exceedingly acute inflammatory infiltration of the submucous tissue at the entrance of the larynx.

At the Clinic at Greifswald a patient (who had come there on account of Bright's disease) died of inflammatory œdema of the larynx a few minutes after entering the institution.

On post-mortem examination a sharp piece of the rib of a tobacco-leaf was found, with one end penetrating the wall of the right *ventriculus Morgagni.* The patient, on his arrival, was sitting in a wagon smoking a cigar.

The extension of inflammatory processes from the neighboring parts to the larynx is also a frequent cause of submucous laryngitis. Thus simple inflammatory œdema may supervene on wounds of the larynx and its vicinity, on retropharyngitis, tonsillitis, pharyngeal diphthèria, angina Ludovici, and parotitis.

But the submucous tissue is by far the most frequently affected through *inflammations of the perichondrium of the laryngeal cartilages*—that is, through those destructive processes that penetrate to the perichondrium, above all, by tuberculous, syphilitic, typhous, and carcinomatous ulcerations. The more slowly these destructions penetrate in depth, and the slighter the inflammatory irritation of the tissues concerned, the more readily is a *chronic* form of submucous inflammation developed, terminating in hard, circumscribed œdema.

Not seldom the *acute infectious processes* are the cause of phlegmonous laryngitis ; this is true of pyæmia and septicæmia, ulcerative endocarditis, typhus, variola, scarlatina, measles, and erysipelas. The pathological connection between these usually very acute forms of phlegmonous laryngitis and the infectious diseases above named is not yet clear. Often enough a high grade of catarrhal inflammation of the laryngeal or pharyngeal mucous membrane may constitute the primary disturbance ; but such an explanation is certainly not sufficient for all cases, especially not for those of ulcerative endocarditis, pyæmia, and erysipelas. Here we must, in part, consider it probable that the excitors of inflammation have been transported, by the way of the circulation, and in part that there was a more independent localization of the infectious process within the larynx, the latter being especially claimed by Ryland, Budd, Watson, Friedreich, Pitha, and others, for those instances of submucous laryngitis accompanying erysipelas of the face.

With regard to the genesis of *non-inflammatory, simple, serous infiltration of the submucosa*, which we wish likewise to study here, this is either developed in hydræmia, as one of the manifestations of general dropsy resulting from acute and chronic nephritis, from malarial cachexia, amyloid degeneration of the kidneys, etc. ; or, as the dropsy of obstructed circulation, resulting from diseases of the heart, emphysema and cirrhosis of the lungs ; or, finally, as the result of circumscribed obstruction in the laryngeal veins, through compression of the superior and inferior thyroid veins ; or, further, of the facial vein, or even the internal jugular and the innominate veins. The œdema will be unilateral or bilateral, according to the site and extent of the hindrances to the circulation. Such compression may be produced by enlargement of the thyroid gland, swelling of the lymphatic and salivary glands, by new formations about the neck, aneurism of the aorta, etc.

As a general rule, dropsy of the soft parts about the laryngeal entrance, the result of hydræmia or obstructed circulation, is not observed as often as the inflammatory serous or sero-purulent infiltration of the submucosa, following inflammations and suppurations in the larynx itself or in its vicinity.

The general experience on this subject seems to be contradicted by the collection of cases which von Hoffmann has made from the post-mortem reports of the Charité Hospital from 1869 to 1871, inasmuch as local diseases were found but ten times in thirty-three cases of laryngeal œdema. They were distributed as follows: phlegmon of the neck, 3; fracture of the thyroid cartilage, 1; laryngeal diphtheria in variola, 1; syphilitic ulceration of the neck, 1; tuberculous ulceration of the larynx, 2; laryngeal ulceration with bed-sores, 2. On the other hand, the 23 cases occurring with general diseases were as follows: with disease of the heart, 9; nephritis, 8; uterine thrombophlebitis, 1; puerperal septicæmia, 1; pulmonary phthisis, 1; pulmonary emphysema, 1; pulmonary emphysema with parenchymatous nephritis, 2. We must remember, however, that the entire number of observations is still too small, and especially that it does not include cases of œdema from wounds, foreign bodies, cauterization, scalding, perichondritis, etc.

As regards the *age* and *sex* of those attacked, the great variety of etiological conditions precludes the establishment of any general principle. We can only say that the larger number of cases is liable to occur during middle age, because the above-mentioned causes of phlegmonous laryngitis are then most frequent, and that the male sex is the most exposed, owing to the numerous injuries incident to the pursuits followed by men. The statistics of Sestier, compiled without any reference to the primary processes, also correspond to this view, as he found the period of greatest frequency of the disease to be between the ages of eighteen and fifty, and that 131 cases out of 187 were in men.

Pathology.

Anatomical Changes.

As a matter of course, the anatomical condition varies—quite independently of the primary affection—according to the intensity, the extent and the duration of the inflammation, and of the œdematous infiltration.

In *diffuse* submucous laryngitis, *running an acute course*, the soft parts about the entrance of the larynx are found more or less uniformly swollen, especially the lax duplicature of the aryteno-epiglottidean ligament, so rich in connective tissue. Next in frequency comes the submucous tissue of the epiglottis or of the false vocal cords, more rarely that of the true vocal cords and of the parietal walls below them. The mucous membrane over the portions infiltrated is tensely stretched and more or less reddened according to the grade of the inflammation. The aryteno-epiglottidean folds, which are especially affected, swell up to great, round, tense, or flabby rolls, which, together with the misshapen, tumefied epiglottis, diminish, to a greater or less extent, the calibre of the laryngeal entrance. The fluid infiltrated is either purely serous, clear and yellowish in color, or more gelatinous, flowing but slowly or not at all on an incision being made into the soggy tissue,[1] or it may be more sero-purulent; or, finally, quite purulent.

Spreading of the inflammatory swelling upwards, to the soft parts of the pharynx, is, as a rule, rare, and when it occurs is usually the result of erysipelas, suppuration of the cellular tissue, etc.

The œdema is very often limited to the aryteno-epiglottidean folds; it is frequently more marked on one side than on the other, which points to a one-sided course of the submucous inflammation.

In rare instances there is an infiltration of the *submucous tissue of the vocal cords themselves* producing stenosis. A very significant case of the kind has been reported by Risch.[2] It is of peculiar interest, too, because the author was permitted to observe the anatomical changes within ten minutes after death, the larynx having been removed for that purpose.

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[1] In twenty-three post-mortem examinations, Sestier saw the fluid flow out freely, without any or with but slight pressure, in ten cases, on an incision being made; in six cases but a scanty amount of fluid was evacuated, on repeated pressure; in seven there was no flow whatever, the material infiltrated being of gelatinous consistency or appearing coagulated.

[2] Berliner klin. Wochenschrift, 1866, No. 33.

The patient, a strong man—formerly a soldier, and now a night-watchman—had been complaining for three days of a simple laryngeal catarrh, when, on returning to an overheated room after a cold night's service, he was soon attacked with slight dyspnœa. This increased rapidly. At eleven o'clock A. M. Dr. Risch found that there was already a high grade of laryngeal stenosis. He drove home in haste to get instruments for tracheotomy, and on his return (fifty-five minutes later) found the man dead. "After my departure," says Risch, "the man moved about, sometimes sitting, sometimes lying down, again going to the window to look for me. Then he said, in a rough voice, which frightened those about him, that I would be too late; soon after that he became very wild, often clutched furiously at his throat, then he leaped up, fell in a heap and began to rattle."

Examination of the larynx, which was immediately removed, showed inflammatory œdema of the epiglottis (the anterior surface), the aryteno-epiglottidean folds, the false and the true vocal cords, evidently resulting from simple catarrhal laryngitis. Two tensely swollen folds, beginning to the right and left of the epiglottis, ran to the arytenoid cartilages, receiving a deep impression from the superior cornua of the thyroid cartilage. These folds were the œdematous aryteno-epiglottidean ligaments, their posterior halves being pressed closely together, so that when they were drawn apart, their internal surfaces were found to be irregularly flattened. The opening constituting the mouth of the larynx measured two-tenths of an inch antero-posteriorly and three-tenths of an inch laterally. The blunt ends of the folds lay barely two-tenths of an inch below the tips of the superior cornua. The bases of the same fully filled the space between the tips of the arytenoid cartilages and the point of union of the superior cornua with the thyroid cartilage; posteriorly they bulged over, and passed into the likewise infiltrated mucous membrane of the arytenoid cartilages, forming at the same time grotesque welts or rolls. From here the submucous infiltration extended into the pharynx as far as to the middle of the posterior wall of the plate of the cricoid cartilage.

In the cavity of the larynx, at each side of the opposing surfaces of the rolls formed by the swollen aryteno-epiglottidean ligaments, there were two tense folds, or rather strips of mucous membrane, extending downwards, as follows: one strip on each side ran down to a very prominent roll, four-fifths of an inch long by three-tenths of an inch broad, which took the place of the false vocal cord. A similar strip on each side ran down to the corresponding true vocal cord, or rather to a roll one inch and one-tenth long by four-tenths of an inch broad, occupying the place of the cord. Of these four rolls, the upper two lay in close contact with one-another, *while the lower two (the swollen true vocal cords) were flattened against one another to the extent of a fifth of an inch (in breadth), thus hermetically closing the passage.*

The color of the rolls which narrowed the mouth of the larynx, as well as of the strips running from here downwards, was a bright yellow; that of the false and true vocal cords, which shone as if varnished, was yellow, with a tinge of green. On being incised, all these rolls were found to contain watery, quite colorless serum. The portions lying between them, most of which were infiltrated, were of a pale rose color. There was no œdematous swelling below the vocal cords; the mucous membrane was reddish and marbled, and covered with a sticky secretion. None of the laryngeal muscles were discolored or infiltrated, but they all showed a good red color and firm fibres.

Evidently, in this case, the œdema of the upper portions had extended rapidly to the vocal cords, and had produced death by suffocation, through the enormous swelling of the latter. This coincides with the fact that the voice was at first clear and afterwards alarmingly rough, and with the sudden death.

Œdema below the cords is no less rare than that of the cords. It was first mentioned by Sestier, was designated as œdème sousglottique by Cruveilhier, and more recently has been again described by Gibb, according to whom, it has also been observed by Russel and others. Extension of the submucous infiltration to the trachea seems to be equally uncommon. Sestier found œdema of the trachea only seven times among one hundred and thirty-two cases of œdema of the upper air-passage (l. c., p. 55).

In *non-inflammatory œdema* there is no injection and swelling of the mucous membrane; the œdematous portions are pale, or pale red, translucent, and flabby.

Phlegmonous inflammation of the submucous connective tissue, terminating in *the formation of circumscribed abscesses*, originates by far the most frequently in perichondritis. Still, rare instances of laryngeal abscess do occur, in which some traumatic injury, over-exertion of the larynx, rheumatic or catarrhal influences must be considered as the cause, or in which no cause at all can be assigned.

The seat and extent of abscess formation is very various. Abscesses are most frequently one-sided, on the outer or inner surface of the cartilaginous framework of the larynx, especially at the base of the epiglottis, on the arytenoid cartilages, in the sinus pyriformis, on one of the plates of the thyroid cartilage or in one aryteno-epiglottidean ligament. The inflammatory swelling of the submucous and mucous tissue is greater or less in degree, according to the extent and acuteness of the process. During the progress of the suppuration œdema of the epiglottis and of the aryteno-epiglottidean folds is frequent enough, and so also is œdematous infiltration of the cellular tissue of the neck, if the abscess is on the outer surface of the larynx. The region corresponding to the seat of the abscess is more or less strongly prominent. The spontaneous escape or the artificial evacuation of the pus occurs, according to the seat of the abscess, outwards or inwards, into the larynx, pharynx, or œsophagus.

The termination of phlegmonous inflammation in *diffuse suppuration* and in *ichorization* seems to be very rare, especially as a primary affection. So far as I know, the only case observed is that of Cruveilhier,[1] which is to be given more in detail further on. Here there was a primary and very extensive suppurative infiltration of the submucous tissue and of the mucous membrane on the aryteno-epiglottidean folds, the epiglottis, the contiguous portions of the pharynx and the base of the tongue, and at some points the mucous membrane was exfoliated. This termination may be somewhat more frequent in acute infectious diseases, where the connective tissue, infiltrated with pus, may undergo ichorous degeneration, and the mucous membrane, over a considerable area, may become necrotic. The infiltration of pus may extend to the submucous connective tissue of the fauces and pharynx, as well as to the inter-muscular and subcutaneous cellular tissue of the neck (Rokitansky). Ichorous degeneration and death are here the rule, and the case of Pollock,[2] in which life was saved by tracheotomy, in spite of the obstacles to success presented by purulent infiltration of the soft parts of the neck, may be regarded as exceptional.

Chronic submucous laryngitis, *chronic œdema*, is almost always a circumscribed affection, limited to certain parts, and is associated with the slow processes of ulceration which penetrate deep to the perichondrium and the cartilage, especially those of a phthisical character, with the insidious forms of perichondritis laryngea circumscripta, and with ulcerative new formations. The favorite seat of this chronic œdema is the arytenoid cartilages (on one or both sides in unilateral or bilateral perichondritis of the processus vocalis), the false vocal cords, etc. The submucous connective tissue is not only infiltrated, but also hyperplastically thickened; so is the perichondrium. The mucous membrane is likewise hypertrophied and more or less injected.

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[1] Anatomie pathologique, Tom. I. Livr. V. Pl. II. Fig. 1.

[2] In Gibb, l. c., p. 235.

Symptomatology.

The manifestations of submucous laryngitis and of simple non-inflammatory œdema vary much, according to the seat and the extent or intensity of the process.

Highly acute diffuse infiltration runs its course under the most stormy manifestations, and may cause death within a few hours, or even minutes, through closure of the laryngeal entrance, if the right help is not afforded at the right time. This highly acute course may be seen in œdema from wounds of the larynx, from foreign bodies that have punctured it, from scalds, and cauterizations. Hyperacute inflammation most rapidly leads to such great swelling of the soft parts about the laryngeal entrance, that the access of atmospheric air to the lungs is very soon restricted to the last degree.

As a prototype of this hyperacute course, Boerhaave's case, as mentioned by van Swieten, is generally cited. A gentleman, who was engaged at a dinner-party, suddenly spoke in an altered voice, which those who were at the table with him took as an evidence of pain. After a few minutes of dyspnœa, he sank to the floor, dead. Post-mortem examination showed œdema of the larynx.

I have had occasion to see several such cases, two of which were seen after death. A laborer, returning from his work very hungry, greedily swallowed his soup, containing chopped-up portions of meat. He was suddenly seized with extreme dyspnœa, and died before I reached him. Post-mortem examination showed œdema of the glottis from a pointed piece of bone, the end of which had thrust itself into the walls of the laryngeal entrance.

A second case—sudden death from œdema of the glottis by spearing of the walls of the laryngeal ventricle with a piece of the rib of a tobacco-leaf—has been given above.

In a third case, that of a young man, seventeen years old, a piercing pain in the throat, with violent desire to swallow, and quickly supervening symptoms of laryngeal stenosis, followed immediately upon the eating of some bread; and the patient came running to me in the greatest distress and with great dyspnœa, believing that he had swallowed a needle with the bread, and that it was sticking in his throat. Laryngoscopic examination showed not only dark red injection of the mucous membrane of the laryngeal entrance, and serious œdema of the right aryteno-epiglottidean fold, but also a little, black, foreign body in the right sinus pyriformis, which, on being extracted with the laryngeal forceps, proved to be a hard, pointed splinter of wood, about a fifth of an inch long. In this case barely fifteen minutes had elapsed from the moment of the entrance of the splinter to its extraction, and yet the inflammatory injection and swelling were already quite considerable.

The observation of Cruveilhier's, referred to above, of acute primary phlegmonous laryngitis terminating in *diffuse purulent infiltration* of the mucous and submucous tissues, properly belongs here.

A man, fifty-six years of age, was taken acutely sick with languor, pain in the neck, and fever. On the third day the pain in the neck was more severe, the palate reddened and swollen, and speech interrupted (venesection). At evening laryngeal dyspnœa supervened almost to the point of suffocation; the voice was hoarse and croup-like, and there was increased swelling of the palatine arches and velum. (Thirty leeches.) On the morning of the fourth day, pain in the neck, dyspnœa and angina had disappeared, and the voice was nearly normal. At evening difficult, whistling respiration returned, with hoarseness and delirium. On the fifth day his condition was the same, and he died at noon.

Post-mortem: The aryteno-epiglottidean folds were enormously swollen, their inner surfaces touching one another and only leaving a narrow passage for air at their posterior portion. These folds, as well as the mucous membrane of the posterior surface of the larynx, the adjoining portions of the pharynx, the base of the tongue and the epiglottis, were of a whitish-yellow color, the mucous membrane everywhere appearing as if saturated with pus, the submucous tissue everywhere infiltrated with pus, which, however, nowhere ran together into an abscess. The mucous membrane was exfoliated at some points; the vocal cords and that portion of the larynx lying below the glottis were quite normal.

Cases of *secondary* phlegmonous laryngitis, running a similar course, resulting from *erysipelas*, are reported by Ryland (l. c., pp. 69 and 73) and by Porter.[1] With reference to the anatomical characteristics and clinical course of laryngitis in erysipelas, an essential distinction will have to be drawn between the laryngitis which occurs in primary, sporadic erysipelas of the face, and that which accompanies the erysipelas sometimes endemic in hospitals, which usually attacks wounds, being malignant in its course, with a tendency to purulent infiltration. The cases reported by Porter from the Dublin hospitals (1835 and 1836) seem to have been of the latter variety. The fever was of an asthenic, typhoid type, the submucous connective tissue of the larynx was infiltrated with gangrenous ichor, and the termination was, in all cases, fatal.

The acute laryngitis, not unfrequently met with in cases of sporadic, primary erysipelas of the head, and which is of but slightly marked phlegmonous character, usually runs a favorable course. The following instance, observed by Türck (Klinik, p. 161), may serve as a type of this catarrhal laryngitis, with moderate œdema, occurring in erysipelas.

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[1] Observations on the Surgical Pathology of the Larynx and Trachea, etc. Dublin.

A woman, forty-six years of age, had erysipelas of the face with difficulty of swallowing, hoarseness, and some dyspnœa, the palatine arches and tonsils being but slightly inflamed. The tissues investing the left cartilage of Wrisberg and that of Santorini were greatly swollen. The visible portion of the left false cord was moderately reddened and swollen, and thereby the anterior and only visible portion of the true cord was partly covered. The left arytenoid cartilage was completely immovable; so, also, was the left vocal cord, which was set somewhat inwards. The posterior surface of the epiglottis as well as the greater part of the remaining mucous membrane of the larynx was only very moderately reddened.

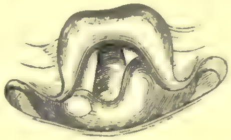

FIG. 1.

Phlegmonous laryngitis in erysipelas of the head. After Türck.

Under the use of ice-pills and the mixtura oleosa the difficulties subsided, and the hoarseness disappeared in the third week. The diminution of the swelling and the return of mobility of the left arytenoid cartilage and left vocal cord followed gradually. On the twenty-fifth day the patient was dismissed. The redness and swelling were still considerable; mobility was entirely restored. On the forty-third day the mucous membrane was quite pale and the swelling was reduced to a minimum.

In these extremely acute cases a chronic laryngeal affection often exists previous to the fatal catastrophe. Violent manifestations of submucous infiltration appear on slight irritation of the larynx; for instance, loud speaking, etc. Often enough a slight degree of œdema may have previously existed without any symptoms, and only be developed to a dangerous extent by the mechanical or other irritation.

An instructive case of this kind, which might find a parallel in any large hospital, is communicated by Ruehle (l. c., p. 157).

A patient, convalescent from exanthematous typhus, had already left his bed, when an abscess appeared under the left sternomastoid muscle. It was evacuated by an incision on the fifth day, the pus being normal. On the afternoon of the same day the patient had visitors, and talked for a considerable time with a loud and clear voice. Suddenly, after having a little while before taken a drink of water, he sank back in his bed, and died before any help could reach him. Post-mortem examination showed notable œdema of the epiglottis and aryteno-epiglottidean folds, as well as of the ventricle of Morgagni, and a somewhat reddened mucous membrane. The cavity of the abscess in the neck was quite empty, the surrounding connective tissue œdematous.

Cases of laryngeal œdema ending thus suddenly in death may also occur among convalescents who are very much reduced in strength, even without any demonstrable cause, and especially without any previous laryngeal affection. This is particularly the case after typhus, during the course of Bright's disease, of tumors of the mediastinum, aortic aneurism, etc. (Consult Sestier and Emmet.[1])

Ruehle (l. c., p. 153) observed a case of intra-laryngeal œdema running a very acute coruse in a patient with aortic aneurism; tracheotomy removed the danger of suffocation, and through the gaping tracheal wound the œdematous rolls above were seen crowding downwards. The canula was removed after thirteen days. In consequence of repeated hemorrhages from the aneurism which penetrated into the œsophagus, the patient died on the twentieth day. Post-mortem examination showed moderate œdema remaining in the ventricle of Morgagni without any other changes in the larynx, and an aneurism of the aorta of the size of a goose's egg, situated behind the point of origin of the left subclavian artery, which had penetrated into the œsophagus two inches below the cricoid cartilage. No light was thrown on the pathogenesis of the alarming laryngeal œdema.

Diffuse submucous infiltration often runs a more *subacute* course, developing slowly during days or weeks, causing no laryngeal stenosis, or only a moderate degree thereof, and often receding again without having produced any symptoms that caused concern. Without laryngoscopic examination this subacute laryngeal œdema escapes diagnosis, and often surprises us, as an unexpected accompanying condition, on post-mortem examination, when death has resulted from the primary affection. Still, as has already been stated, such subacute infiltration not rarely develops under very slight irritation to the highest grade of swelling and laryngeal stenosis.

2. *The circumscribed form of phlegmonous laryngitis, which leads to abscess formation*, sometimes runs its course very acutely within a few days, sometimes more slowly, especially when it has its seat on the external surface of the larynx. The seat, intensity and extent of the inflammatory œdema, following the

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[1] Amer. Jour. of Med. Sciences, July, 1856.

progressive abscess formation in the submucous or subcutaneous cellular tissue, essentially determine the picture which the disease is to present. If a laryngeal abscess is developed about the entrance of the larynx, the picture of acute laryngeal stenosis may appear so suddenly and be so predominant that nothing but laryngoscopic inspection can determine with any certainty the point of origin of the disease.

As a general rule, suppurative, circumscribed, phlegmonous laryngitis in the interior of the larynx runs its course with a moderate fever, a pricking pain at a definite spot, which is increased by the act of swallowing and by pushing the larynx about, and a moderate disposition to cough. Laryngoscopic examination shows a circumscribed arching of the abscess walls, with strong injection of the mucous membrane over it and in its vicinity; touching the abscess with the laryngeal sound increases the pain. If the abscess is seated immediately above or below a vocal cord, the movements of the latter cease. As the abscess approaches its period of rupture, the pain, and especially the dyspnœa, is increased through the growing œdema of the soft parts about the laryngeal entrance. The rupture of the abscess, if death has not previously supervened through stenosis, or relief been afforded by an incision, is accompanied by strong manifestations, a sense of suffocation and retching. With the escape of pus the storm is speedily quieted.

The seat of the abscesses reported in the cases scattered throughout literature is very varied. They have been found at the base of the epiglottis (Döring), at the free end of the epiglottis (Lewin, Tobold), on one arytenoid cartilage (Gottstein, Schnitzler), on the left half of the cricoid cartilage (Türck), in the aryteno-epiglottidean folds (Tobold), in the ventricle of Morgagni (Berger).

If there is great œdema of the surrounding parts, it may be very difficult to arrive at a *diagnosis;* and even when we succeed in reaching and evacuating the collection of pus, the question of whether the abscess owes its origin to primary, phlegmonous laryngitis, or to perichondritis, usually remains unsettled.

Those laryngeal abscesses formed on the *outer surface of the larynx* (the thyroid and cricoid cartilages) are in general of much less serious character. Still, the case reported by Ruehle (l. c., p. 167), in which an abscess on the outer surface of the left plate of the thyroid cartilage led, previous to its opening, to œdema of the larynx (which, it is true, rapidly subsided after evacuation of the pus), shows how serious the disturbances may be, even here.

Circumscribed, chronic, submucous laryngitis—chronic œdema—which, as stated above, is associated with deep ulceration penetrating to the perichondrium, especially to that of the arytenoid cartilages, either causes the patient no subjective annoyances, or gives him the sensation on swallowing of having a foreign substance in his throat, or causes fits of strangling to accompany swallowing, portions of fluid passing into the larynx, owing to imperfect closure of its entrance. The situation of things is first revealed by a laryngoscopic examination. The region of one arytenoid cartilage and corniculum (seldom of both) is uniformly swollen, the swelling is pale, pale-blue, or pale-red, and feels tolerably resistant on being touched with the sound. The ulcer lying at the foundation of the chronic œdema is often not to be discovered, if it is seated on the inner surface of the arytenoid cartilage, immediately over the vocal process. On post-mortem examination we often find a very small ulcer, from which the probe passes in to the necrotic spot on the arytenoid. These chronic submucous swellings or œdemas persist, quite unchanged, for months; they may slowly increase in area, seldom diminishing. In some cases they terminate, especially on acute exacerbations of perichondritis or necrosis of the cartilage, in acute submucous infiltration presenting the picture of the most violent form of laryngeal œdema.

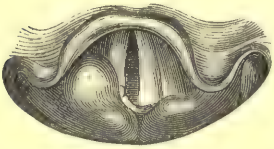

FIG. 2.
Chronic laryngeal œdema.

Analysis of the most Important Symptoms.

The symptom which is usually the first to appear, and soon crowds all others into the background, is *laryngeal dyspnœa*, the prevention of inspiration by the laryngeal stenosis, and especially by the longitudinal rolls or welts which lie in the way and are formed of the aryteno-epiglottidean folds, or by the irregularly swollen epiglottis. This laryngeal dyspnœa is at first generally inspiratory alone, while the expiratory stream of air passes the larynx without any difficulty. This inspiratory character of the earlier dyspnœa depends upon the valve-like pressing inwards and against one another of the aryteno-epiglottidean folds by the pressure of the column of air from without, whereby they are also pressed against the false vocal cords and the epiglottis; whereas the expiratory stream of air pushes them apart and towards the rear, thus leaving the passage free. The mechanism of this action, which was first demonstrated by Lisfranc on the body of a person who had died of laryngeal œdema, is, however, of more theoretical than practical interest, as the purely inspiratory character of the dyspnœa does not last long, but is soon associated with expiratory difficulty, too, owing to the increased swelling of the aryteno-epiglottidean folds rendering them motionless, as well as to the swelling of the epiglottis and superior cords. Furthermore, inspiratory dyspnœa, while it lasts, is by no means characteristic of œdema alone, and therefore cannot determine the diagnosis.

Laryngeal stridor varies in intensity according to the grade of swelling of the soft parts. In the highest grades of narrowing of the glottis, the stridor can be heard afar off; and the other symptoms of laryngeal stenosis also reach their highest intensity, such as the inspiratory descent of the larynx, sinking inwards of the suprasternal fossa and the epigastrium, decreased frequency and increased depth of the inspirations, etc. The flapping valve sound which Legroux claims to have heard in inspiration was probably somewhat theoretically constructed on the above-mentioned valve theory, and may perhaps have been caused by some tenacious secretion. It is evidently of no significance. In the lungs, as in all high grades of laryngeal stenosis, no vesicular murmur can be heard, but only the sound produced in the obstructed larynx.

An exhaustive diagnosis can only be made by means of *laryngoscopic* and *digital examination.* They determine the degree and the seat of swelling, and the question of the presence, the site, and the composition of a foreign body, or an abscess.

Laryngoscopic examination demands great skill and quickness, as the patient cannot long maintain the necessary position of the head and mouth for an examination, on account of the great dyspnœa. The masses of mucus that gurgle forth also greatly interfere with the view—as is the case in all high degrees of laryngeal stenosis—less so, to be sure, before tracheotomy than after it, because afterwards the inspiratory and expiratory stream of air, which moves the foamy secretion away, is no longer present.

The mirror shows the soft parts of the laryngeal entrance, and especially the aryteno-epiglottidean folds, the superior cords, and the epiglottis, as being swollen out of all shape. The aryteno-epiglottidean folds resemble rolls or welts, which touch one another in the median line and are displaced inwards. The color of the mucous membrane is deep red or pale red, according to the intensity of the inflammation, or it may even be quite pale (perhaps because the pressure of the infiltrated fluids compresses the vessels, or because there is œdema without inflammation), and the tension of the swollen parts varies, in a corresponding degree, from a highly tense, stretched condition to a relaxed flabby state.

Some typical images from Türck's "Klinik" may here find place.

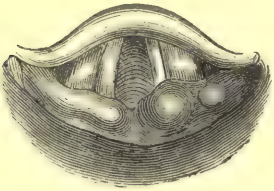

FIG. 3.

Traumatic laryngeal œdema. After Türck.

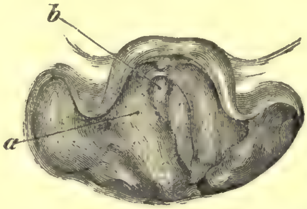

FIG. 4.

Extensive phthisical ulceration of the larynx. A high degree of laryngeal stenosis from œdema. *a*, Right aryteno-epiglottidean fold. *b*, Anterior division of the right vocal cord. After Türck.

In the absence of a mirror, or when it is absolutely impossible to examine with the mirror, one may inspect the upper portion of the epiglottis (seldom more), when the tongue is well drawn out and movements of retching are caused by pressing the spatula on to the base of the tongue.

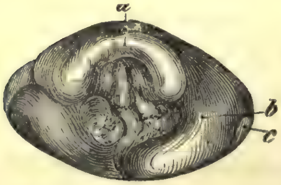

FIG. 5.

Phlegmonous laryngitis with phthisical ulcers. *a*, Epiglottis. *b*, Left aryteno-epiglottidean fold. *c*, Left sinus pyriformis. After Türck.

Digital examination, in the majority of instances, gives information at least with regard to the condition of the epiglottis. If the larynx lies high, and the examiner has a long forefinger, he will be able also distinctly to palpate the aryteno-epiglottidean fold.

The *voice* is either normal or to a greater or less degree hoarse, up to the point of complete loss of tone. The *cough* is like a smothered bark, or noiseless, and the single acts of coughing are interrupted by loud long-drawn stridor. An annoying desire to swallow troubles the patient, and, on swallowing, the swelling at the laryngeal entrance makes on him the impression of a foreign body.

The remote effect of the insufficient breathing on the circulation, the nervous system, etc., naturally varies according to the degree of laryngeal stenosis. When but a moderate amount of stenosis exists, dyspnœa arises only on movements of the body, or sometimes in paroxysms when the body is completely at rest, without any apparent cause that might produce them. When the amount of stenosis is greater, dyspnœa is permanent, and is still further increased by accumulations of mucus in the laryngeal entrance, by swallowing, speaking, etc. Patients now assume the standing or sitting posture, their arms resting on their knees or on a table, and breathe with the greatest effort; sometimes they walk up and down in the greatest unrest. The pulse is frequent, exceedingly small and soft. The skin is pale, cool, covered with sweat, the lips livid, the face turgid, the eyeballs protruding, and the conjunctiva injected with venous blood.

Terminations and Prognosis.

The *termination* of phlegmonous laryngitis may be favorable when the grade of its development is slight and the primary inducing cause has ceased. The submucous infiltration, if it is serious in character, is then quickly reabsorbed; if it is purulent, it may break through the mucous membrane. We cannot determine at present whether this favorable termination is frequent, as there has not been enough investigation made into the frequency of the lighter forms of œdema. Examination with the mirror is generally not undertaken until laryngeal dyspnœa supervenes, and the light forms, which present no symptoms attracting the attention of patient or physician, have probably, hitherto, been commonly overlooked. The increasing value attached to the use of the mirror will tend to throw light on this subject too ; and, as the experience of the past few years shows, we shall often discover the lighter cases, if we accustom ourselves, in all those affections which may lead to œdema, to use the mirror early, and without waiting for urgent symptoms, on the slightest appearance of pain, hoarseness, etc. The frequency with which a slight or moderate degree of œdema is found, upon post-mortem examination of persons who, during life, showed no striking symptoms referable to the larynx, leads us to believe that slight œdema, capable of resolution, is more common than has hitherto been supposed.

The higher grades of laryngeal stenosis, due to submucous infiltration, usually terminate in *death*, if timely interference does not prevent. In such cases death is either produced by suffocation, the laryngeal stenosis rapidly advancing to complete closure of the glottis, or by carbonic acid poisoning, due to the gradually increasing interference with the entrance of air into the lungs. In the latter instance death is preceded by prolonged coma, which, as in croup, is at first interrupted by severe fits of dyspnœa, until at last all reaction ceases, and paralysis of the heart and the respiratory muscles closes the scene. Although statistical tables are of very little value, in view of the many heterogeneous processes which the older authors classed under the name of "œdema," yet a general idea of the frequency with which a fatal termination follows may be gathered from the statement of Sestier, who counted one hundred and forty deaths among one hundred and sixty-eight cases of œdema of the glottis.

The termination of diffuse purulent infiltration is no doubt usually unfavorable; ichorization of the purulent infiltration probably always ends in death.

These general considerations, and due regard to the special circumstances of the individual case, above all, to the pathogenesis and the acuteness of the process, determine the *prognosis*. We must bear in mind, as a general rule, that in severe cases the danger to the life of the patient, if the physician maintains an expectant attitude, is uncommonly great, and that even postponing tracheotomy for a few hours may be destructive of the patient, if the physician leaves him in the meantime. The fact is, there is no estimating the rapidity with which stenosis of the glottis may advance.

The case of Risch, which was given above (p. 798) is very instructive in this respect. Quite a similar incident came to my knowledge. A certain man, a scholar of some note, who was suffering from suppurative retro-pharyngitis, was seized with œdema of the glottis. The symptoms of laryngeal stenosis increased rapidly to so alarming an extent that the physician who was called in, a well-known surgeon, found immediate tracheotomy indicated; and hastily ran home to obtain the necessary instrument. But here, too, death occurred within fifteen minutes and before the physician had returned.

We should make it a rule, under no circumstances to leave a patient with laryngeal œdema, and, if the instruments are not at hand in time, to perform tracheotomy with a penknife rather than let the patient suffocate. This was done by a physician with whom I am acquainted, who was making a journey across country on the island of Rügen, and, being called into a farm-house to see a patient with œdema of the glottis, found himself without even a pocket-case.

The instance which Hughes (l. c.) narrates is also a very pretty illustration of this. A student of medicine saved a man, who was at the point of suffocation from œdema of the glottis, by cutting through the crico-thyroid membrane with his penknife, and introducing the tube of his penholder as a canula.

It is often impossible, in view of the acuteness of the process and the pressing need for performing tracheotomy, at once to arrive even at an approximately reliable diagnosis and prognosis. This must be reserved for a more quiet time, after the operation. On the other hand, when the œdema develops gradually it is possible, by calling to one's aid all the subjective and objective signs, to be in the clear as to diagnosis and prognosis, and to base one's therapeutic measures thereon.

Treatment.

In the treatment of phlegmonous laryngitis and of laryngeal œdema, circumspection and presence of mind, on the part of the physician, are more than ordinarily requisite. As the greatest danger to life may arise at any moment, it is important that every preparation should early be made for the worst contingency.

Otherwise we must proceed according to the nature of the process lying at the foundation of the evil, and remove every injurious influence which might aggravate a moderate œdema. With reference to the last point, speaking must be strictly prohibited, and the greatest care must be exercised to maintain the air of the room at a uniform temperature, and to keep out dust, smoke, etc.

As regards medicinal and operative treatment, the attempt should be made, as long as laryngoscopic examination reveals moderate swelling, to reduce the swelling by antiphlogistics and astringents. For this purpose a strong solution of nitrate of silver (from fifteen to twenty-five grains to the ounce) may be applied once a day with a brush or sponge ; between times inhalations of atomized tannin solution, alternated with solutions of bromide of potassium and of morphine may be used, as well as alum gargles. In addition to this, according to the indications of the individual case, a moderate abstraction of blood may be employed, cold or moist-warm applications to the neck, the frequent swallowing of pieces of ice, energetic derivation to the intestinal canal (saline cathartics) and to the cutaneous surface of the lower extremities (stimulating foot-baths).

If, in spite of all this, the dyspnœa increases, we must unhesitatingly proceed to *scarification* of the œdematous portions, carried on under the guidance of the mirror (or, in case of need, using the left forefinger as a guide). This operation, first performed by Lisfranc, was afterwards employed and urgently recommended by the best authors, such as Sestier, Valleix, and others. Its performance has unquestionably been rendered far more easy of late through the assistance of the mirror, and may be performed, with the laryngeal knife, by any physician at all accustomed to laryngoscopy. Several long incisions are to be made, whereupon the swelling generally collapses at once.

The opening of submucous laryngeal abscesses is undertaken in the same manner, and only presents greater difficulties if they are deeply situated, upon or below the vocal cords.

An unsuccessful result following scarification, or the attempt to open a laryngeal abscess, is an indication for the performance of *tracheotomy*, and the physician may now under no circumstances leave his patient until the danger of suffocation is removed.

The use of the *nail of the index finger*, cut to a point, to incise the œdematous swelling, as recommended by Legroux, was no doubt well meant, theoretically, but can hardly have any practical result. There is equally little prospect that the complicated mechanism of simultaneous scarification and compression, as recommended by Sestier in his "Pressoscarificateur" (a sort of long forceps with four sharp knife blades), will ever be adopted in practice, inasmuch as twenty-five years have elapsed since its recommendation without any further observations being reported in its favor.

Catheterism of the larynx, recommended by Thuillier and by others more recently, is likewise impracticable, because it is very difficult to introduce the catheter through the œdematous parts; then it is so intensely irritating that it can only be tolerated for a short time; and on its removal it must leave the œdema aggravated.[1]

After the performance of tracheotomy, the treatment of the laryngeal affection is to be continued according to the indications presented in each individual case. As a matter of course, the removal of the canula and healing of the wound is not to be undertaken until repeated trials of letting the patient breathe while the artificial opening is closed have proved the full and permanent sufficiency of the passage at the laryngeal entrance.

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[1] Compare *Hüttenberger*, Ueber den Katheterismus des Larynx bei der croupösen oder diphtheritischen Erkrankung desselben. Jahrb. der Kinderheilk. N. F. VIII. 1874, p. 89.

PERICHONDRITIS LARYNGEA.

Inflammation of the Perichondrium and its Results.

Aside from the appropriate chapters in the clinical works already cited of *Albers*, *Ryland*, *Porter*, *Trousseau* and *Belloc*, *Andral*, *Ruehle*, *Friedreich*, *Türck*, *Tobold*, and the anatomico-pathological works of *Hasse*, *Cruveilhier*, *Rokitansky*, *Förster* and *Rheiner*, the following special works are to be consulted: *Flormann*, Von einer in Vereiterung übergehenden Halsentzündung. Sammlung auserlesener Abhandlungen. Leipzig. 1791. Bd. XIV.—*Mohr*, Beiträge zur pathologischen Anatomie. Kitzingen. 1838.—*Albers*, Ueber einige Krankheiten der Kehlkopfknorpel. *Graefe* und *Walther's* Journ. für Chirur. und Augenheilk. Bd. XXIX. 1840. —*Dittrich*, Prager Vierteljahrschrift. Bd. XXVII. 1850. S.—*Pitha*, Prager Vierteljahrschrift. Bd. I. 1857. S.—*Türck*, Ueber Perichondritis laryngea. Allgem. Wiener med. Zeitung. 1861. No. 50. —*Theopold*. Ueber Perichondritis laryngea im Anschluss an Typhus. Diss. Jena. 1867.—*Poignon*, De la périchondrite laryngée. Thèse. Paris. 1869. —*Retslag*, Ueber Perichondritis laryngea. Diss. Inaug. Berlin. 1870.—*Morell Mackenzie*, Transactions of the Pathol. Society Vol. XXII. pp. 46 u. 56. 1871.—*Bösensell*, Ueber Perichondritis laryngea. Diss. Inaug. Berlin. 1872.—*Schech*, Zur Casuistik der Perichondritis laryngea. Bayr. ärztl. Intelligenzblatt. 1872. No. 23.—*Gerhardt*, Laryngologische Beiträge. D. Arch. f. klin. Med. Bd. XI. 1873. S. 578.—*Schroetter*, Laryngologische Mittheilungen (Jahresberichte). 1871. S. 24 und 1874. S. 13.—*Krishaber* and *Lepine*, A case of necrosis of the cricoid cartilage. Annales de l'Oreille et du Larynx. Tome II. No. 1.—*Barie*, A case of general necrosis of the laryngeal cartilages. Bulletin de la Société Anatom. de Paris, 1875, p. 238.

Etiology.

Inflammation of the perichondrium of the laryngeal cartilages, and of the substance of the latter, may arise from traumatic causes, and perhaps also from rheumatic influences, as well as from over-exertion of the vocal organs; still the instances of primary disease of the perichondrium—aside from those of traumatic origin—are, in general, rare. Much more frequently inflammation of the perichondrium is the result of the deeper diseases of the mucous membrane of an inflammatory or destructive character, especially of tuberculous, typhous, syphilitic or cancerous ulcerations. Albers (l. c.) claims that the perichondritis results from a primary *laryngeal chondritis*, which latter is supposed to be especially induced by early ossification of the laryngeal cartilages.

The primary development of perichondritis is, however, more strongly emphasized by recent authors. Türck and Schroetter (l. c.) in particular have communicated a number of cases in which, on the most careful study of the history and condition, no other primary affection could be discovered. I have also observed two cases of perichondritis in which neither during life nor on post-mortem examination was there any occasion found for the development of the inflammation. For that matter, Flormann, towards the end of the last century, made public three cases of perichondritis of the cricoid cartilage in which he could find no other etiological circumstance than the continued loud crying of the patients, who were boatmen.

Dittrich counts among the traumatic causes of perichondritis the pressure of the ossified cricoid cartilage against the vertebral column. He draws a parallel between the inflammatory disturbance of nutrition which is thus developed in the perichondrium of the posterior surface of the plate of the cricoid bone, and bedsores developed over the sacrum in typhus fever, etc.

The conception of the process, as being analogous to that of bedsores, would find considerable support in the fact of the occurrence of perichondritis arytænoidea in typhus, and of typhous ulcers between the arytenoid cartilages. The result of the modern treatment of typhus also speaks in its favor. Just as bedsores on the sacrum, so also have ulcers and perichondrial inflammations in the larynx become exceedingly rare since the introduction of methodical antipyretic treatment, so that now, even in hospitals abounding in typhus [exanthematicus], many months pass without a case of the kind being seen.

We must not fail to mention, in this connection, that perichondritis cricoidea may be induced by the frequent introduction of the œsophageal sound in old persons whose cricoid bone is ossified. I have seen such a case, and believe that they are not as rare as one might suppose from the silence maintained on this point in literature. At all events, advanced age and great prominence of the cricoid cartilage demand great care in the introduction of the sound. As soon as symptoms of irritation appear, the introduction of the sound must be discontinued for a considerable time.

As regards the *age* and *sex* of those attacked, as well as the nature of the primary diseases, a table of twenty cases, which Retslag has collected from the reports of post-mortems of the Berlin Pathologico-anatomical Institute, shows that sixteen out of this number were men and four women. In the matter of age, the third decennium was the most liable to attack. In all cases the inflammation was secondary, and the original diseases (which, also, induced death in all cases) were distributed as follows: tuberculosis, ten times; typhoid fever, eight times; pleuritis ulcerosa, once; leptomeningitis and chronic myelitis, once. The seat of the affection was on the cricoid cartilage, eleven times; the thyroid cartilage, three times; the cricoid and epiglottis, twice; the cricoid and arytenoid, once; the thyroid and arytenoid, once.

Pathology.

Pathological Anatomy.

The seat of the inflammatory changes is generally on the cricoid cartilage or on one arytenoid cartilage. If the course is acute, the inflammatory swelling is soon followed by purulent infiltration which, before the tense tissue of the perichondrium is fully reached and perforated, dissects it loose from its cartilage, whereupon the latter becomes partially or entirely necrotic. Great inflammatory œdema of the surrounding submucous connective tissue may, at this stage, produce stenosis of the laryngeal entrance.

If the course is slower, the abscess forms a protuberance beneath the perichondrium, inwards (into the cavity of the larynx) or outwards (for instance, towards the pharynx), or in both directions, which, of itself, as well as through the œdema of the surrounding connective tissue, may cause serious hindrance to respiration. If the pus is slow in breaking through, or its artificial evacuation is long delayed, the cartilage concerned becomes totally necrotic, softens and breaks down into fragments of a dead, dirty-yellow or blackish-brown color, which are enveloped in pus or ichor. The perichondrium is entirely destroyed. The wall of the abscess is infiltrated with pus and subsequently thickened, being either fatty or callous.

If the evacuation of the abscess through the softened perichondrium and the mucous membrane finally takes place with a great rent, the little arytenoid cartilages may be driven out as a whole, while of the larger cartilages (cricoid and thyroid) necrotic pieces only may be loosened and floated out. If an entire arytenoid cartilage is expelled, the abscess collapses after the complete evacuation of the pus, and may become closed by new formations of connective tissue. But the result of the defects in the cartilaginous framework is always to produce such changes in the position of the soft parts as make themselves known by a narrowing of the calibre of the larynx, and by serious disturbance of the functions of the vocal cords.

This process of healing, which, however, is always rare, may also take place after the loss of single portions of cartilage, whereupon the perichondrium remains thickened by a sort of rind of connective tissue. The expulsion of an entire large cartilage—for instance, the plate of the cricoid cartilage—is an extremely rare occurrence, and recovery after such an occurrence as was observed by Hunter[1] is still more rare.

In the majority of instances of perichondritis of the thyroid or cricoid cartilage, the necrotic cartilage remains in its position after the opening of the abscess, maintains a profuse suppuration, and causes callous thickening of the connective tissue. The abscess often breaks outwards and inwards at the same time, and thus fistulous tracks are formed from the cavity of the larynx to the pharynx, or the outside of the neck.

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[1] Compare Ruehle, l. c., p. 175.

In one of the two cases of primary perichondritis which I alluded to above, the left plate of the thyroid cartilage had become necrotic, and a complete laryngeal fistula had resulted. Colored fluids which I injected into one of the external openings reached the cavity of the larynx, excited cough, and were expectorated.

The process may persist for a long time at this stage. The cartilage may become gradually dissolved by softening, or may crumble, or it may become ossified and then carious, or finally it may become so far increased in volume by new formations of cartilage or of bone as to close the cavity of the larynx, as observed by Gintrac.

Perichondritis of the epiglottis is generally very rare, and then usually occurs in connection with the same process in the cricoid and thyroid cartilages, or with ulcerative processes. According to Rokitansky, inflammation of the epiglottis leads "to the breaking down and absorption of the cartilage and its substitution by a hard, shrivelled, half bony cicatricial tissue. It is at the same time diminished in size and misshapen; section thereof shows pale, rotten remnants of cartilage."

Symptoms and Course.

Laryngeal perichondritis is probably seldom recognized as such in the beginning. On the one hand, the manifestations of the secondary inflammation of the perichondrium are masked by the symptoms of the primary affection; on the other hand, even in a primary case, the initial symptoms of perichondritis are susceptible of such varied significance that a positive diagnosis—at least at first—is generally impossible.

Circumscribed *painfulness* of a cartilage, increased by pressure on the larynx, or by pushing it about (in affections of the cricoid or thyroid cartilage), or increased by speaking, and swallowing (in affections of the arytenoid and the plate of the thyroid), as well as *cough*, *hoarseness*, *swelling of the lymphatic glands* of the neck, should at once cause us carefully to examine the larynx by means of the mirror, etc., if any disease of the larynx is present, or other disease, which experience has taught us may stand in a causal relation to perichondritis (typhus, variola, syphilis, pulmonary phthisis). Careful examination and watching of the patient will become absolutely necessary as soon as *symptoms of laryngeal stenosis* are added to the above manifestations, even though but to a slight degree, inasmuch as an increase of the stenosis, up to the point of suffocation, may take place suddenly and within a few hours.

If the abscess in the perichondrium is not artificially opened, it may happen that, while the raising and thinning of the mucous membrane over the abscess progresses, the purulent infiltration in the submucous connective tissue spreads, or the abscess may remain quite circumscribed. In either case, it is seldom possible to distinguish, with certainty, between an abscess of the perichondrium and a primary submucous abscess. The acute swelling of the surrounding soft parts preceding spontaneous rupture of an abscess inwards—which has its analogue in the inflammatory œdema of the neighboring parts, in a tonsillar or retropharyngeal abscess—when it takes place within the larynx or at its entrance, almost invariably causes the highest grade of laryngeal stenosis and danger of suffocation unless speedy and energetic measures are taken against it. It happens but seldom that the abscess breaks in the midst of severe suffocative attacks, and that, with the evacuation of the pus, which is not rarely mingled with fragments of cartilage, the alarming symptoms of laryngeal œdema quickly subside. Generally, the danger of suffocation compels the most speedy resort to tracheotomy.

The laryngoscopic image found[1] varies very much, according to the seat of the inflammation, its extent, the intensity of the collateral œdema, and the participation of the muscles in the purulent infiltration. The acute swelling has nothing characteristic about it; it is sometimes more strongly developed behind, sometimes in front, or laterally.[2] Under some circumstances paralysis of the muscles of the vocal cords may be of significance as bearing upon the diagnosis—for instance, in perichondritis of the plate of the cricoid cartilage—inasmuch as paralysis of the posterior crico-arytenoid muscles, caused by purulent infiltration, must lead to inspiratory stenosis of the glottis. In a case of perichondritis arytænoidea of the left side, in which, during life, the vocal cord remained immovable in the median line, Schech was able to satisfy himself, by post-mortem examination, that the left posterior crico-arytenoid muscle was pale and disseminated with tuberculous masses, and that, furthermore, the processus muscularis was detached from the left arytenoid cartilage, which was denuded of perichondrium all about it.

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[1] The first examinations with the mirror in perichondritis were undertaken by Türck, who indeed deserves great credit for all he has done to extend our knowledge in this direction.

[2] Compare the full reports of cases by *Türck* (Klinik, p. 207); by *Schroetter* (Laryngol. Mittheilungen, 1874, p. 13, et seq.); and by *M. Mackenzie* (l. c.).

After the abscess has broken through, laryngoscopic examination is generally able more sharply to determine the seat of the perichondrial affection, judging by the point of exit of the pus, and the now more circumscribed prominence of the parts concerned. The results of destruction, the position and mobility of the soft parts for laryngeal respiration, etc., can also now be more accurately observed.

It will but seldom be possible, as is described by Böscnsell, to see the perichondrial ulcer through the laryngo-tracheal wound, and to be able to clean it of shreds of mucous membrane and necrotic fragments of cartilage. (In this case the cricoid cartilage and the crico-thyroidean membrane were also split.)

The peculiarities of the different pictures of disease, according to the localization of the perichondritis on the several cartilages, which have been thoroughly studied by Türck, are as follows:

Perichondritis arytænoidea is brought about especially by phthisical ulcers on the posterior wall of the larynx, most frequently single ulcers on the vocal process, or on the anterior side of the arytenoid cartilage and the cartilages of Santorini, these ulcers penetrating to the perichondrium and laying bare the cartilage. In contrast to their depth, they are often very small in circumference, and on making a post-mortem one needs a probe in order to demonstrate the necrosis of the cartilage. These ulcers often escape a diagnosis, entirely or in part, owing to the inaccessibility of their position, on the posterior wall of the larynx, to the mirror. If one is unable, by means of the lateral position of the mirror, and rotation of the head (Türck), even to see the edges and indentations of the ulcers, he must fall back, for his diagnosis, on the chronic inflammatory œdema of the soft parts about the arytenoid cartilages and those of Santorini and Wrisberg, as, in phthisical persons, this pretty certainly points to arytenoid perichondritis. The immobility of the arytenoid cartilages is of significance, although it proves nothing alone, as it may be caused by anchylosis of the crico-arytenoid articulation, as well as by extensive submucous exudation, or by paralysis of the muscles or of the recurrent laryngeal nerve.

If one arytenoid cartilage has been entirely eliminated, the soft parts, with Santorini's cartilages, sink in (compare Figs. 8 and 9), if the dense infiltration of the soft parts does not prevent it, and the median line is pushed over to the diseased side. The movements of the sound arytenoid also appear somewhat limited, on account of the falling away of the arytenoid muscle. The elimination of a necrotic arytenoid cartilage, however, does sometimes take place without any considerable disturbance, and the appearance of alarming laryngeal œdema is not always necessary.

Perichondritis cricoidea, whether occurring primarily or secondarily, is of far greater clinical significance than perichondritis arytænoidea, or thyroidea alone. It occurs principally on the plate, seldom on the sides or anterior portions of the bone, and, especially when it is caused by phthisical ulcers, is often combined with necrosis of one or both arytenoid cartilages, as the process generally originates on the upper lateral portions of the plate, and passes from here over on to the arytenoid cartilages. Hereby the articulation is naturally either destroyed or at least anchylosed. Often enough the disease may travel in the opposite direction, from the arytenoid to the cricoid cartilage.

The *symptoms* of primary perichondritis of the cricoid, aside from pain and difficulty of swallowing (particularly when the posterior surface of the plate is affected), are especially laryngeal stenosis, caused by the protuberance of the abscess on the posterior wall, which may, within a few days, rise to the highest grade, and the displacement of the vocal cord of the affected side, or of both vocal cords, towards the median line (in perichondritis of the lateral portions, or of the posterior surface of the cricoid plate, through paralysis or destruction of one or both posterior crico-arytenoid muscles).

The case described by Pitha[1] presents a very instructive example of perichondritis cricoidea.

A strong man, thirty years old, was seized, according to the statements made, immediately after taking a cold drink while heated, with rigors, heat, difficulty of swallowing and of breathing, a painful little cough, and headache. In forty-eight hours the laryngeal dyspnœa had reached such a degree that tracheotomy had to be performed during the night, and it was with difficulty that the arrested breathing could be re-established. Severe pain on swallowing continued. Double pneumonia followed, and death on the seventh day of the disease.

Post-mortem examination showed no œdema of the laryngeal entrance, but obliteration of the calibre of the larynx by the protuberance of its posterior wall. The partially ossified cricoid cartilage was detached from the surrounding soft parts through purulent infiltration, and was in part necrotic. On the left wall of the thyroid cartilage, too, the intensely red mucous membrane was detached and thrown into folds. In the same way the mucous membrane of the trachea was of a dirty, dark-red color, with numerous little collections of pus, from the size of a millet-seed to that of a hemp-seed, scattered through it, and in some places detached from its cartilaginous rings. The veins of the thyroid gland and at the bifurcation of the trachea contained thrombi. The connective tissue at the latter point was partly infiltrated with serum and partly with ichor. There was double lobular pneumonia.

Türck has drawn the laryngoscopic picture of a similar though chronic case (occupying about three months) of *primary perichondritis of the plate of the cricoid cartilage and the left half of its arch, with anchylosis of the left arytenoid*, the abscess remaining closed (Klinik, p. 212; compare Fig 6).

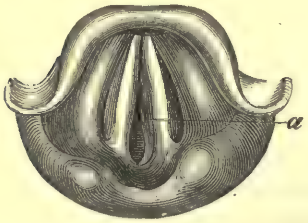

FIG. 6.

Perichondritis cricoidea.
a, Inner wall of the abscess, presenting itself as a longitudinal fold or welt. After Türck.

In this case (in which tracheotomy was not performed) post-mortem examination showed the covering of the left (anchylosed) arytenoid cartilage and the left aryteno-epiglottidean fold to be œdematous, the chink of the glottis narrowed, the cricoid cartilage surrounded by an abscess containing tough, viscid, yellowish pus, its plate necrosed, and the upper portion of the same surrounded by the very much thickened and ossified perichondrium as by a shell. The anterior division of the left half of the cricoid as well as the left half of the first tracheal cartilage was melted down to fragments nearly as thin as paper. The collection of pus arches inwards into the interior of the larynx on the left side, and sinks downwards between the trachea and thyroid gland. There was pneumonia of the left lower lobe.

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[1] Beitr. z. Würdigung d. Tracheotomie, etc. Prager Vierteljahrschrift, 1857, Bd. I.

I had quite a similar state of things lately in a man with subacute perichondritis of the cricoid of the left side (compare Fig. 7). Eventually the pus emptied itself, after the wall of the abscess had become somewhat pointed, and the patient left the hospital improved.

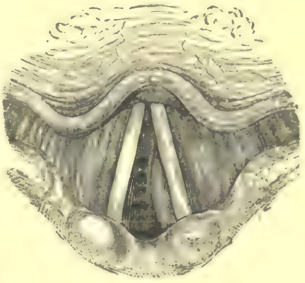

Fig. 7.

Perichondritis cricoidea. Arching forward of the walls of the abscess under the left vocal cord.

Further instructive observations and laryngoscopic images have been furnished by Türck with regard to *perichondritis of the plate of the cricoid with loss of the left arytenoid and collapse of the soft parts*, all due to tuberculous ulceration.

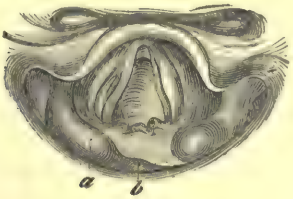

Fig. 8.

Perichondritis cricoidea.
a. Right cartilage of Wrisberg
b, Right cartilage of Santorini. The left cartilage of Santorini is sunken in. After Türck.

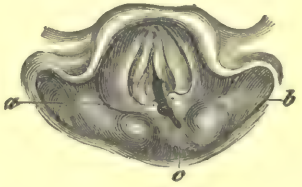

Fig. 9.

The same case, eight weeks later.
a and *b*, Indentations; and
c, Posterior end of the edge of the ulcer visible on the anterior surface of the posterior wall of the larynx. After Türck.

At first there was no stenosis of the glottis (Fig. 8). Eight weeks later there was serious stenosis (Fig. 9), produced by the collapse of the soft parts on the posterior wall, following the loss of the left arytenoid cartilage and the cartilage of Santorini, widening and ulceration of the vocal cords, and the pushing of the right cartilage of Santorini toward the left, across the median line.

Post-mortem examination (compare Türck's Klinik ; Figs. 93 and 94, pp. 250 and 251) revealed a large ulcer on the posterior wall of the larynx. The upper border of the cricoid was partly lost. The arytenoid and cartilage of Santorini on the left side were missing, the right arytenoid laid bare on its internal surface and superficially necrotic.

Finally, we give place here to one more laryngoscopic picture of Türck (Klinik, p. 220), which was obtained from a perichondritis of the cricoid, principally of the right side, with loss of the right arytenoid, occurring in the fourth week after an attack of typhoid fever. The case was that of a man thirty-three years of age, the subject of tabes; the image represented was taken in the sixth week of typhoid fever and about the second week of the perichondritis.

At the anterior region of the right arytenoid, an irregularly round, nodulated, red tumor, of the size of a pea, covered with yellow spots, stood out, and could not be regarded as anything else than an abscess. It prevented a view of the posterior division of the right, true vocal cord. The covering of the right cartilages of Wrisberg and Santorini and the arytenoid cartilage, as well as the right aryteno-epiglottidean fold, was very greatly swollen and reddened. The cartilages just named, as well as the right vocal cord, which was set inwards, and the anterior visible portion of which was but slightly reddened, were immovable. The movements of the left vocal cord and its cartilage were normal, only that the movement outward seemed to be somewhat restricted. Amongst the other manifestations were: severe attacks of laryngeal dyspnœa, moderate pain in the cricoid (spontaneous, on pressure, and on swallowing), and hoarseness. During the last days, grayish, purulent sputa, and fetid breath. Death followed during about the fourth week of the perichondritis.

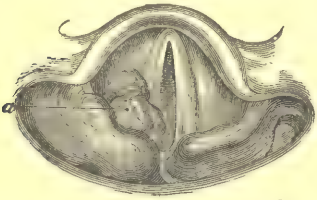

FIG. 10.
Perichondritis cricoidea et arytænoidea dextra. *a*, Wall of abscess. After Türck.

On post-mortem examination, the larger posterior half of the cricoid cartilage was found denuded of perichondrium, surrounded by a collapsed abscess partly filled with ichorous, fetid pus. This abscess had perforated the mucous membrane almost immediately behind and below the ventricle of Morgagni, therefore on the inner surface of the lower division of the arytenoid cartilage, making an opening of the size of a pea, surrounded with necrotic blackish-gray edges. The plate of the cricoid was split into two halves from above downward, the left half, being to a great degree preserved till near the median line, only showing superficial losses of substance, while a very considerable portion of the posterior and upper part of the right half, as well as the entire right arytenoid cartilage, was lost. The left arytenoid was well preserved, only the surface of the articulation with the cricoid was roughened, and its ligamentous apparatus was, to a great degree, destroyed, in spite of which the arytenoid was still quite in its normal place. The other conditions found were typhous ulceration of the intestines, lobular pneumonia, and pulmonary œdema.

Perichondritis thyreoidea—perichondritis of the thyroid cartilage—may be limited to the inner surface, and lead to stenosis by bulging of the abscess walls and œdema of the surrounding submucous tissue. Not rarely, however, it establishes itself, at the same time, on the outer surface of the cartilage, and ends with perforation of the abscess outwards.

The *diagnosis* in *perichondritis thyreoidea interna* is based on the circumscribed painfulness of one thyroid cartilage, the more or less rapidly increasing manifestations of laryngeal stenosis, the visible bulging into the calibre of the larynx, and, finally, the breaking through of pus and, perhaps, the history of the case.

In *perichondritis thyreoidea externa*, besides the unfailing pain, a swelling of the affected region appears, which, if diffuse, may obliterate the prominences of the larynx and give to the front of the neck a cylindrical form, but which may also be restricted to single portions of the thyroid—for instance, to the pomum Adami or one lateral wall—and may round off the part concerned in a somewhat misshapen manner. As the swelling increases, a doughy œdema is the forerunner of fluctuation and pointing of the abscess. After the latter is opened it is not difficult, by means of the probe, to inform one's self to some degree with regard to the extent of the cavity of the abscess and the necrosis of the cartilage.

If a true *laryngeal fistula* has arisen, by perforation of the abscess outwards and inwards, the point of the probe may pass into the cavity of the larynx, and here be demonstrated with the laryngeal mirror, as was done in one case by Schroetter. In a case of this kind, in which the ossified thyroid cartilage was felt to be rough to the probe, I succeeded in having a colored fluid, which I injected into the external fistulous opening, reach the cavity of the larynx, and be expelled therefrom by the cough which was immediately excited.

Terminations and Prognosis.

The most usual termination of perichondritis is in *death*, as, on the one hand, the primary affections lying at the foundation of it are most unfavorably influenced by the supervention of perichondritis, and, on the other hand, when this affection itself is primary, the destruction of the cartilages and the prolonged suppuration may exert a deleterious influence upon the constitution. Still, recovery is a more frequent method of termination in primary perichondritis than in that induced by tuberculosis, syphilis, typhus, or variola. Complete or partial recovery may take place with elimination of necrotic pieces of cartilage and the replacement of the same by a fibrous structure. These latter cases are the ones most frequently resulting in changes of position and disturbances of function in the soft parts of the laryngeal entrance and the upper laryngeal cavity, caused by the above-mentioned defects in the cartilaginous framework and the shrinking of the newly formed fibrous tissue, whereby laryngeal breathing and the formation of the voice are so far interfered with that the tracheal canula cannot be removed nor the tracheal wound closed, and voice-formation is either entirely destroyed or at least materially impeded. The elimination of one arytenoid cartilage, with its corniculum, is enough to produce this result. Of course the consecutive disturbances are much more considerable if a part of the plate of the cricoid is lost at the same time. The pictures furnished by Türck (in his Atlas, Plates V. and VI.) give instructive illustrations of this. The smallest degree of, at least, immediate danger is presented by perichondritis thyreoidea. Still, even here the final termination is generally unfavorable.

It hardly needs to be stated that appropriate medical interference, especially the timely performance of tracheotomy, has a material influence on the result. The majority of cases that terminate in recovery owe it to timely laryngo-tracheotomy. It is only by the opening of a new air-passage, in general, that the possibility for the slow repair of the inflammatory destructive processes in the larynx is given.

The development of general *emphysema of the skin* from a perforating ulcer of the cartilage appears to be a very rare occurrence. I find in literature only a general statement in Rokitansky[1] that emphysema of the skin may follow perforation of the larynx by ulcers, and a case by Wilks,[1] which happened in a perichondritis following typhus.

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[1] In the first edition of his Handbook of Pathological Anatomy.

A boy, twelve years old, who was brought into Guy's Hospital with a severe attack of typhus, had an emphysematous swelling on the neck appear on the twelfth day, which spread rapidly over face, breast, and arms. Ten days later he died. The post-mortem showed a perforating laryngeal ulcer at the posterior commissure of the vocal cords. A probe introduced through the opening, which was about the size of a pea, reached to the left and downwards into a cavity between the trachea and œsophagus, filled with muco-pus. Evidently the air had escaped from here into the posterior mediastinum, and had thus reached the subcutaneous cellular tissue.

The great rarity of these cases induces me to cite a similar one from my own practice.

Margaret Grimm, aged four, was admitted to the Poliklinik at Erlangen on the 10th of August, 1863, suffering from a severe attack of typhus. The disease had already existed for several days, and ran its course with very high fever (104° to 105.8° in the rectum), continued unconsciousness, involuntary stools, a fetid coating of the mouth, much cough, pneumonic consolidation on the right side, below. On the night of the 22d to the 23d (about the middle of the third week of the disease) an emphysematous swelling appeared in the jugular region of the neck, and in the course of the next few hours spread upwards, on the left side, to the border of the lower jaw and to the mastoid process; downwards, over the trunk to the crest of the ileum; on the right side it extended over all the triangles of the neck and below the clavicle as far as the second rib. Wherever the connective tissue was loose, as in the suprasternal and subclavicular regions, over the false ribs and the abdominal muscles, the emphysema formed great bags of air. On the posterior walls of the chest the emphysema was developed to but a limited degree on both sides, and was entirely absent over the spine. During the next few days the emphysema extended on the left side to the symphysis pubis, and on the flexor surface of the arm to the hand; on the right side, over the anterior wall of the chest to the edge of the ribs. At the same time parotitis of the left side was developed. There was no hoarseness, no appearance of laryngeal stenosis. Death ensued on the 28th of August (about the beginning of the fourth week of the disease).

Post-mortem on the 29th of August (Prof. Zenker). Body much emaciated. Subcutaneous emphysema of the neck, chest, and belly, on both sides. On opening the thorax, the connective tissue of the mediastinum appeared filled with air-bubbles; the same were also found at the root of the left lung and under the left pulmonary pleura. On the most powerful blowing of air into the lungs (*in situ*, by means of a tube tied into the trachea) they become excessively distended, but no air escapes anywhere; neither does air escape on making strong pressure on the distended lungs, the trachea being closed. Moderate bronchial catarrh and croupous pneumonia of the right lower lobe.

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[1] Transactions of the Pathol. Society, 1858, Vol. IX. p. 34.

The source of the subcutaneous emphysema was found in the larynx, in the form of *an ulcer, of the size of a lentil, at the base of the left arytenoid cartilage, under the left vocal cord, penetrating to the necrotic arytenoid and cricoid cartilages.* At the base of the right arytenoid there was an erosion.

Aside from this, there was a typhous spleen and swelling of the mesenteric glands. In the cæcum, ascending and transverse colon, there were abundant typhous ulcerations; in the ileum, reticulated patches; the parotid was the seat of multiple abscess.

After the most careful examination of the thoracic viscera, and especially insufflation of the lungs from the trachea, had shown that the subcutaneous emphysema could not have originated from the lungs, the bronchi, or the trachea, the only point of origin left was the perforating laryngeal ulcer. Probably the severe cough, which in this case was maintained by an active bronchitis, contributed to the result, the air, which was subjected to severe pressure by the paroxysms of coughing, having escaped outward, into the subcutaneous cellular tissue, through the ulcer that lay beneath the closed glottis.

Treatment.

The treatment, at the beginning, especially in primary perichondritis, must be antiphlogistic and derivative—leeches to the neck, the application of a narrow ice-bag to the larynx, swallowing pieces of ice, the inunction of pustulating ointments to the neck, the insertion of a seton.

If stenosis of the larynx supervenes, we must proceed according to the cause of the stenosis, as shown by laryngoscopic examination—scarifying the œdema of the soft parts about the laryngeal entrance, if present, or opening a protruding abscess with the laryngeal knife. If the latter does not succeed, or if the other attempts to lessen the stenosis (poultices to the neck, the inhalation of atomized warm fluids, emetics) do not have the desired effect, we must proceed to tracheotomy before carbonic acid poisoning sets in, in order to guard against the disturbances otherwise caused in the lungs (lobular pneumonia, etc.), by prolonged stenosis of the upper air-passages.

As a curiosity, the case of Fyffe deserves mention, in which tracheotomy was necessary twice within a short time. A soldier had perichondritis (probably syphilitic) of the cricoid and thyroid cartilages, and tracheotomy was performed during a suffocative attack. Seven days later, a black, fetid piece of bone was coughed up, which seemed to come from the thyroid cartilage. Thereupon recovery and closure of the tracheal wound followed. Three weeks later there was a relapse of the perichondritis and a second tracheotomy.

The local treatment of the laryngeal mucous membrane with mild astringents, the inhalation of bromide of potassium solutions, with or without morphine, may, under some circumstances, have a favorable influence on the stenosis, especially if large ulcerated surfaces exist; but their effect is generally transitory, and probably but seldom averts the need of tracheotomy.

Abscesses on the outer walls of the larynx, originating from the thyroid and cricoid cartilages, are to be opened early.

Perichondritis cricoidea may, by compression of the pharynx, as well as by extension of the suppurative process to the submucous tissue of the lateral and posterior pharyngeal wall (Dittrich, Mackenzie), occasion such interference with swallowing and such strangling, that nourishment by means of the œsophageal tube becomes necessary (Mackenzie). The mechanical irritation of the laryngo-pharyngeal affection, by the introduction of the œsophageal tube, can of course only be unfavorable.

If, after the favorable termination of the perichondritis, *laryngeal stenosis* remains, as is usually the case, the attempt must be made, by systematic methods of dilatation, gradually to widen the passage to such a degree that the canula can be removed and the tracheal wound healed. The hope of the spontaneous disappearance of stenosis is almost always illusory;[1] the patients are doomed—if the stenosis is not mechanically removed—to wear a canula as long as they live. It needs no argument to convince us that this is very distressing to the patient, not only by reason of the annoyance associated with the removal, cleaning, and reintroduction of the canula, but, as Schroetter very justly remarks, particularly on account of the alarm and inquisitive questioning which the sight thereof causes among the uninitiated. The improved laryngo-therapeutic measures of our day can point to many admirable results, in this particular, and their number would certainly be much greater if the labors of the laryngeal surgeon were not often made futile by the lack of energy and persistence on the part of the patients. These go from one specialist to another, but remain with no one long enough to arrive at a permanent result.

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[1] According to Theopold's statistics, in twenty-two cases of perichondritis after typhus, tracheotomy was successful eight times, so far as the preservation of life was concerned. In none of these eight patients, however, did the stenosis afterwards diminish to such an extent as to permit of the removal of the canula.

The methods of dilatation are, in brief, as follows:

1. *Dilatation by the stream of air of inspiration and expiration.* The fenestrated canula is closed externally by a cork, and left closed until evidences of insufficient breathing appear.

Gerhardt[1] has reported a case cured in this way. The changes found, eight weeks after tracheotomy, consisted in two ridges under the vocal cords which only left a narrow, somewhat oblique slit between them, swelling and partial immobility of the left arytenoid cartilage, and a small defect and afterwards granulation sproutings at the base of the left vocal cord. The treatment at first (from May, 1869) consisted in the introduction of a sound into the larynx through the tracheal wound, afterwards in periodical closure of the canula. After the patient had, in the meantime, gone through with an attack of pneumonia, and the condition of things in the larynx, especially the mobility, had gradually improved, the patient was able, in February, 1871, to lay aside the canula, and in June of the same year—therefore two years after the beginning of treatment—the tracheal fistula was closed by freshening its edges.

2. *Widening of the stenosis by means of dilators and bougies* has repeatedly been recommended, and various instruments have been constructed for that purpose; for example, one, on the principle of Heurteloup's percuteur (Gerhardt), and others on that of the laryngeal forceps (Navratil, Schroetter, and others). In general, little is to be expected from the dilators with arms, as they irritate too much and cannot be long retained in the larynx. On the other hand, according to more recent observations, and especially to the experiments of Trendelenburg, von Bruns, Oertel, and especially of Schroetter,[2] the introduction of

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[1] D. Archiv f. klin. Med. 1873, Bd. XI. p. 578.

[2] Laryngologische Mittheilungen. II. 1874, p. 32; und Beitrag zur Behandlung der Larynx-Stenose. Wien, 1876.

smooth, hard bougies, of increasing thickness, is to be recommended. It is true that under the use of his tin bougies—which he leaves in position for many hours—Schroetter found that, if he proceeded rapidly with them, severe pain in the neck and head, febrile movements and even new attacks of perichondritis and œdema followed ; still these disturbances afforded no ground for interrupting the treatment while the tracheal wound was open. Granulations in great quantity crowd into the canal that is being dilated, but are best removed by the introduction of the bougies. The parts gradually resume their normal mobility, and, what is most striking, do not lose their normal sensitiveness by mechanical treatment.

Schroetter's results, according to his latest publications, are so brilliant, and their number is already so considerable, that there can scarcely longer be any doubt as to the practicability of this method of dilatation.

If, however, systematic dilatation should not accomplish its end—that is to say, should not open a sufficient way through the larynx for breathing—resection of the larynx would still be in reserve for the worst cases, with the subsequent introduction of an artificial vocal organ, according to Heine's method, which will be more particularly spoken of hereafter, under the head of the Operative Treatment of New Formations.

ULCERATIONS AND TUMORS OF THE LARYNX.

1. Phthisis Laryngis. Consumption of the Larynx.

Petit, De phthisi laryngea Dissertatio. Montpellier. 1790.—*Sauvée*, Recherches sur la phthisie laryngée. Paris. 1800.—*Laignelet*, Sur la phthisie laryngée. Paris. 1806. *Schönbach*, De phthisi laryngea. Dissertat. inaug. Wilnae. 1808.—*Papillon*, Du larynx et de la phthisie laryngée. Paris. 1812.—*Sigaud*, Recherches sur la phthisie laryngée. Strassbourg. 1819.—*Sachse*, Beiträge zur genaueren Kenntniss der Luftröhren- und Kehlkopfschwindsucht. Hannover. 1821.—*Trousseau et Belloc*, Traité de la phthisie laryngée. Paris. 1837.—*Barth*, Mémoire sur les ulcérations des voies aëriennes. Archives général. 1839.—*Louis*, Recherches anat., pathol. et thérapeut. sur la phthisie. Paris. 1843, p. 51, et seq.—*Cless*, Beiträge zur Pathologie der Tuberkulose. Archiv für physiol. Heilkunde. III. p. 596. 1844. —*Neumann*, Ueber die chron., ulcerative Laryngitis. Journ. für Kinderkrankheiten. 1847.—*Rheiner*, Ueber die Ulcerationsprocesse im Kehlkopf. Virchow's Archiv. Bd. V. 1853, p. 534, ff.—*Toulmouche*, Études cliniques sur les ulcerations du larynx et de la trachée artère. Archives générales. 1857. July, Aug.—*Villemin*, Du tubercule. Paris. 1862.—*Virchow*, Die krankhaften Geschwülste. Bd. II. p. 644. 1865.—*Ter Maten*, Nederlandsch Tijdschrift voor Geneeskunde. Treede Afdeelig. 1865, p. 36.—*Prinz*, Ueber Larynx-Affectionen bei Tuberkulose. Archiv der Heilkunde. IX. 1868.—*Schnitzler*, Ueber Kehlkopfgeschwüre mit bes. Rücks. auf ihre laryngoskopische Diagnose und locale Therapie. Wiener med. Presse. No. 14, ff. 1868.—*Norton*, Ulcers of the larynx simulating phthisis. Brit. Med. Journ. Jan. 22, p. 78. 1870.—*Mandl*, Ueber Kehlkopfschwindsucht. Wochenschrift d. Ges. d. Wiener Aerzte. No. 47. 1870.—*Schroetter*, Oestreich. Zeitschrift f. prakt. Heilkunde, p. 51. 1870; und dessen laryngologische Beiträge (Jahresbericht). 1871, p. 56, und 1874, p. 72.—*Sommerbrodt*, Ueber die Abhängigkeit phthisischer Lungenerkrankung von primären Kehlkopfaffectionen. Archiv f. experimentelle Pathologie. Bd. I. p. 264. 1873.—*R. Meyer*, Der gegenwärtige Stand der Frage von der Kehlkopfschwindsucht. Correspondenzbl. der Schweizer Aerzte. 1873. No. 13.—*M. Mackenzie*, A lecture on laryngeal phthisis. Medical Record. Nov. 15, 1873.—*Lincoln*, Laryngeal

phthisis. The New York Medic. Record. Feb. and March, 1874.—*Schech*, Die Affectionen des Kehlkopfes in ihren Beziehungen zur Phthise. Bayr. ärztl. Intelligenzblatt. No. 25. 1874. *Marcet*, Tubercular laryngitis. Lancet. Feb. 27, 1875.—*Sawyer*, Tubercular laryngitis. Lancet. Jan. 30, 1875.—*Bucquoy*, On tubercular and syphilitic laryngitis. Gazette des Hôpitaux. April 20-29, 1875.—*Bertolet*, Tubercular ulceration of the larynx and trachea. Philadelphia Med. Times. June 19, 1875.—*Porter*, Laryngeal phthisis. St. Louis Med. and Surgical Journal. Jan. 1875.—*Glasgow*, Laryngeal disease in its relation to pulmonary phthisis. Philad. Med. Times. July 10, 1875.—*Koch*, On laryngeal phthisis. De La Haye. Paris, 1875.—*Starr*, Tuberculous laryngitis. Philadelphia Med. Times. Dec. 25, 1875.—*Sawyer*, Notes on phthisical laryngitis. British Med. Journal. April 15, 1875.—*Isambert*, On miliary tuberculosis of the pharynx and tuberculous infiltration of the epiglottis and larynx. Annales de l'Oreille et du Larynx. No. 3. Tome I.—*Chamberlain*, Laryngeal phthisis. Proceedings of Connecticut Medical Society. 1876.—Compare also the handbooks of pathological anatomy of *Cruveilhier*, *Hasse*, *Rokitansky*, *Foerster*, and *Rindfleisch;* furthermore the works of *Laënnec*, *Ruehle*, *Türck*, *Tobold*, *Duchek*, *Mandl*, *Cohnen*.

Pathogenesis, Etiology, and Pathological Anatomy.

By laryngeal phthisis we understand only those ulcerations of the larynx following pulmonary consumption, and which are caused by miliary tuberculosis and by tuberculous (scrofulous) inflammation. The state of ferment in which the entire doctrine of tuberculosis and phthisis is involved, makes it appear more desirable, on account of their intimate clinical relations, to group together, under a common clinical head, those various different anatomical processes that are here concerned. The clinical designation is by no means prejudicial to the final establishment of the nature and significance of the individual pathological processes lying at the foundation of the clinical picture. On the same grounds Türck chose the still more general designation of "laryngeal diseases standing in connection with pulmonary tuberculosis."

This is not the place to enter into a discussion on the essence of the processes lying at the foundation of phthisis in general, and especially on the role which tubercles and tuberculous or scrofulous inflammation play in pulmonary consumption; we must rather refer to the more detailed discussion of this subject in Vol. V. and one of the later volumes [Birch-Hirschfeld's article on Scrofula] of this Cyclopædia. Here only those points will be considered that are of value in understanding the processes which take place in the phthisical larynx.

As is well known, Laënnec's teaching of the purely tuberculous nature of laryngeal phthisis early met with opposition (Louis, Cruveilhier, Trousseau), and in the latest times the opposition has gone so far as utterly to deny that tubercle is the foundation of laryngeal phthisis. On the other hand, the first authorities in pathological anatomy, Rokitansky, Foerster, and, above all, Virchow, contend for the laryngeal tubercle—in fact, Virchow just exactly recommends the larynx as the most appropriate place for the study of true tubercle.

According to Virchow,[1] the laryngeal tubercle belongs to those which, being especially exposed to external injuries through their superficial position, very easily break down on their surface, and produce little, shallow, simple tuberculous ulcers, without ever becoming cheesy or giving rise to any considerable tumor. This circumstance might explain the striking fact that cheesy tubercles are so seldom found in the larynx either during life or on post-mortem examination—a fact which has always been insisted on with emphasis by the opponents of laryngeal tubercle.

Foerster[2] expresses the same opinion as Virchow, that the tubercle proceeds from the connective tissue of the mucous membrane, is therefore very superficially situated, quickly breaks through outwards, and forms little ulcers. Foerster is of the opinion that doubt with regard to the existence of true tubercle and tubercular ulcers in the larynx, can only result from the lack of thorough microscopic examinations.

Other observers have been able to demonstrate, with certainty, during life, the existence of miliary tuberculosis. Ter Maten has done this in two cases; Türck in one case, in the vicinity of tuberculous ulcers;[3] Schech in the laryngeal muscles (left posterior crico-arytenoid) associated with perichondritis. I myself have had the opportunity to examine two such undoubtedly tuberculous eruptions within the larynx, and find the declaration of Virchow to be evidently correct, that the absence of cheesy tubercles and the shallowness of the ulcers depend on the superficial position of the tubercles and their consequent tendency to decay. It is true that this does not remove the difficulty of demonstrating the tuberculous origin of the ulcers; neither can this difficulty be removed, from a clinical point of view. The pathologico-anatomical character of the base and the border of the ulcer is the only thing that is conclusive on this point.

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[1] Krankhafte Geschwülste. II. 2, p. 645.

[2] Handbuch der pathol. Anatomie. II. Aufl. 1863, p. 320.

[3] Klinik der Kehlkopfkrankheiten, p. 372, and the illustrations in its Atlas. Plate XV. Fig. 5.

Among twelve cases of laryngeal ulcers in pulmonary consumption, Rheiner could in no instance find the characteristic signs of crude cheesy tubercle. On the other hand, he found, here and there, indications of the fresh tubercular granulations of the mucous membrane, also referred to by Hasse, mingled with little round ulcers of the same size, but with flat, non-infiltrated edges, which in all probability had proceeded from those nodules, without however having retained the evidences of their origin.

Rindfleisch, too, virtually confirms the existence of this state of things, inasmuch as he states that he very commonly finds roundish collections of cells, of the size of a glandular acinus, at the base of phthisical laryngeal ulcers, at some distance from the surface, in the midst of connective tissue which is still intact. These collections he can, at present, only interpret as being miliary tubercles. It is true their number is so small, and they are such insignificant new-growths in comparison to the inflammatory infiltration of the actual surface of the ulcers, that Rokitansky is disposed to look upon them only as a sort of pledge of the connection of that process with constitutional tuberculosis. The greatest importance that might be attached to them is that of permanent irritants tending to produce inflammation, and the obstinacy and tendency to relapse, which are peculiar to those catarrhal inflammatory conditions, might be referred back to them.

If, according to this, we cannot doubt the occurrence of miliary tubercles and true tubercular ulcers in the larynx, that does not settle the question of the frequency with which they occur as over against phthisical ulcerations that do not proceed from miliary tubercles.

If we consider, first, the histological changes which are offered by the latter class, we have presented to us, as the *second form, follicular ulcerations.* These have, of late, been most thoroughly studied by Rindfleisch,[1] and have been characterized by him in the following manner:

The ulcerations in the larynx as well as the trachea begin at the mouths of the mucous glands. At first the formation of pus exists in the excretory ducts of the glands as a muco-purulent secretion—the pus is produced in the subepithelial connective tissue of the excretory duct of the gland and poured into the latter; very soon, however, it degenerates into pus formation with loss of substance, into ulceration. A circular, flat, funnel-shaped ulcer arises, with a narrow, but intensely yellow border, by which it is sharply distinguished from the surrounding hyperæmic mucous membrane. In the middle of the loss of substance, the deepest point of the floor of the ulcer is formed, either by the widened excretory canal or the gland itself, or—after suppuration of the body of the gland—by a round cavity corresponding to it. The original characteristics of the ulcer are not obliterated until, in its further course, it extends laterally as well as in depth. By the confluence of contiguous ulcers, for example, the grape-shaped outlines appear, which have generally been looked upon as characteristic of tubercular ulcers—in fact, the grape-shape is particularly pretty here and more distinctly marked than in tuberculous ulcerations of the intestines. The advance of the floor of the ulcer in depth is especially favored by suppuration of the bodies of the mucous glands. A purulent inflammation of the enveloping and interstitial connective tissue of the gland leads to the decay and dissolving of the acini; the entire gland melts away; and when we remember that the mucous glands of the air-passages do not lie in the mucous but in the submucous tissue, we understand why just these ulcers most rapidly induce deep-seated destruction. In fact, we very soon find the floor of the ulcer in the vicinity of the cartilaginous rings or of the laryngeal cartilages, and herewith a new field of disturbance is opened.

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[1] Lehrbuch der patholog. Gewebelehre. IV. Aufl. 1875, p. 325.

According to this representation of Rindfleisch's, then, the primary condition in this form of phthisical ulcers, aside from the scrofulous or tubercular diathesis, would be a *purulent follicular catarrh*, which only gives occasion to deeper alterations under the influence of that diathesis. It is partly the abundant inflammatory, new cell-formation in the subepithelial connective tissue, partly the vulnerability of the latter and of the epithelial covering of the follicles, which leads first to the breaking down of the excretory duct of the gland and gradually to the purulent melting down of the entire gland body.

A similar account of the pathogenesis of follicular ulceration had already been given by Rheiner; he, however, supported by the authority of Louis, as well as by the works of Hastings and Horace Green, associates this alteration less with pulmonary consumption than with chronic pharyngitis (Clergyman's Sore Throat, Hastings). His description of the processes is as follows:

In the interior of the individual glandular sacs a considerable proliferation of cell elements takes place, whereby they are largely distended. The tender connective tissue lying between them is thinned, the walls of the same keep drawing nearer together and form very thin division walls, which finally burst, so that the single compartments unite to form one common cavity, and their contents flow together in a larger collection, which is surrounded by the common connective-tissue envelope, and forms a prominence rising above the level of the surrounding mucous membrane. At their apex there is a little yellowish-white point, and on pressure a drop of a similarly colored, creamy fluid escapes, which sometimes no longer contains any formed elements, but only presents a finely granular, molecular mass. It is a little abscess, whose cover finally bursts through thinning, whereupon its contents are evacuated, and the beginning of an ulcerating surface remains, whose outlines are formed by a thickened ring.

This description of Rheiner's, as well as his statement with regard to the seat of these ulcers, "corresponding to the principal seat of the glands, especially at the base of the epiglottis, extending to the upper vocal cords and along the anterior surface of the arytenoid cartilages, never on the border of the true vocal cords, but a few lines below them, and from there down into the trachea," justifies the conclusion that Rheiner's follicular ulceration is probably etiologically identical with that described by Rindfleisch, since laryngoscopic examination has shown that, in the chronic pharyngitis of the writers above referred to, there is indeed chronic catarrh of the larynx and pharynx, and eventually also hypertrophy of the glands, but extremely seldom any ulceration.

A *third* form of phthisical ulcer seems to proceed neither from degenerated tubercles nor from ulcerated follicles, but from a specific *infiltration of the subepithelial layer of the mucous membrane with cells and nuclei.* Rheiner already described this infiltration of the mucous membrane with little round cells and nuclei, which, when occurring to but a slight degree, is confined to the subepithelial stratum, but when more largely developed causes a circumscribed swelling of the mucous membrane, conceals the tissue elements thereof, and is surrounded by a distended network of blood-vessels. On section the swollen tissue of the mucous membrane appeared of a grayish-red color, almost like marrow, and similar to the typhous infiltrations; still he could find no glandular organs whatever as the starting-point of this cell-proliferation. Ulcers gradually establish themselves upon these infiltrated regions, whose form and depth depend essentially upon the histological structure of the subjacent parts.

According to Rheiner, it is particularly the elastic stratum that for a long time resists the progress of destruction, and this is especially effective in the true vocal cords. If the elastic layer is finally also destroyed, this is very little likely to take place transversely, but rather by means of the separation of the bundles of fibres that are united longitudinally, and thus interstices are formed lengthwise, which keep growing broader and deeper through the successive casting-off of filaments. Thus the beginning and more superficial ulcerations of the vocal cords almost always possess an *elongated flat form*, not that uniform, roundish or ragged configuration, with a tendency to the formation of funnel-shaped depressions, which is to be found in the softer parts where the mucous membrane does not possess that wealth of elastic elements nor their arrangement in bands, and destruction is not limited in any direction. In the cavity below the vocal cords as well as in the trachea, where the arrangement of fibres is more perpendicular, the ulcers, according to Rheiner, show a form more elongated from above downwards, and it is not till the elastic layer is destroyed that they assume uniform dimensions and even show a tendency to extension laterally, when the destruction, for instance, has reached the muscular layer on the posterior wall of the trachea, the fibres of which we know run in a transverse direction.

Rheiner also makes a successful attempt to explain the local predilection of the ulcers when he credits it to the mechanical influences, the pressure, the distortion, to which the mucous membrane is unceasingly exposed during the functional activity of the larynx. The pressure and the distortion which the tips of the vocal processes and the inter-arytenoid region undergo during phonation, and which the cartilages of Santorini and the border of the aryteno-epiglottidean bands experience, through the epiglottis, during deglutition, are certainly of the greatest consequence as sources of irritation. In view of the great vulnerability of the tissues in scrofulous and tubercular organisms, and the specific tendency of the same to react, on comparatively slight irritation, with abundant cell-infiltration and incomplete retrograde metamorphosis, the mechanical causes above mentioned suffice to excite an inflammatory cell-proliferation in the mucous membrane, with secondary destruction through caseation of the cell-membrane.

A *fourth* form, the most superficial and flattest of the phthisical ulcers, has long been distinguished from those penetrating more deeply under the name of *aphthous* or *erosion ulcers*, and the anatomical and clinical peculiarities of the same have been thoroughly established.

The fact that fresh, flat ulcers with a gray floor, and of very great extent by reason of confluence, are very frequently found, amongst consumptives, in the trachea and larynx, alongside of evidently older and deeper losses of substance in the larynx, has of course always attracted the attention of pathologists. They were regarded in part as being tuberculous and identical with the deeper ulcers, only of more recent date, and as having appeared but shortly before death; or as being *ulcers from corrosion or infection*, called forth by the irritating influence of the ichorous secretion, proceeding from cavities, on the superficial layer of the mucous membrane, loosened by long-continued catarrh.

Louis, who first sought to establish this latter view, based it principally on the fact that these ulcers were chiefly found on those parts on which, by reason of the anatomical construction of the upper air-passages and the posture of the patient, the secretions remained longest in contact with the mucous membrane, viz., on the posterior and lateral walls of the trachea, on the vocal cords, especially their posterior commissure, and, finally, on the inferior surface of the epiglottis. Rheiner maintained a very reserved position over against this etiological conception, but, with regard to histological relations, called attention to the fact that, in the neighborhood of these "aphthous" ulcers, whose margins showed no trace of elevation or infiltration, one might, indeed, sometimes find little flat elevations, but that these seemed to be formed by a serous exudation into the most superficial layer of the mucous membrane—at least, that no infiltration of formed constituents into the tissues of the mucous membrane could be demonstrated. The superficial throwing off of the constituents of tissue seems sometimes to originate from these serous exudations, sometimes from the mouths of the glands.

Later observers confirm these results in general, especially the absence of miliary tubercles and of cell-infiltrations at the base and in the neighborhood of the ulcers. Some have gone so far as to regard the ulcers as catarrhal erosions or ulcers without any specificity. To this I cannot agree. These flat, numerous ulcers are just peculiar to pulmonary phthisis — Trousseau already declared that he had seen them only in phthisical subjects —whereas they are not developed even in very old bronchial catarrhs. A direct local relation to the cavities is also not seldom very distinctly demonstrable. In making post-mortem examinations of phthisical patients, in whom ulcerative degeneration was exclusively confined to one superior lobe, I have repeatedly seen the flat ulcers extend from the main bronchus of the superior lobe, through the main bronchus of the lung of that side, to the trachea and the larynx, while not an ulcer could be found in any of the other bronchi. Such observations force us to accept the doctrine of an infectious and destructive effect of the secretion of the cavity on the most superficial layer of the mucous membrane, although the questions of wherein this lies and why it does not appear in all phthisical patients, still remain to be answered. The possibility that these aphthous ulcers may, after all, proceed from decaying miliary tubercles is by no means excluded by the observations made thus far. Foerster[1] does not hesitate to declare that, according to his investigations, most of the so-called aphthous ulcers are really tuberculous, though some rest upon a flat diphtheritic exudation. Rindfleisch, too, on the one hand, emphasizes the infectious quality of the products of scrofulous-tuberculous destructive processes, rich in cells and detritus, and, on the other hand, the tendency, produced through the mechanical lesion, to the formation of erosions, which constitute true *points of inoculation.*

As we have seen above, attention was called by Rheiner already to the mechanical irritations of the mucous membrane, which are especially effective just in the larynx, and their significance in the production of inflammatory irritations, with cell-infiltration and ulcer-formation in the naturally vulnerable mucous membrane, is insisted on by most recent authors. In reality it is those parts most exposed to mechanical irritation in the acts of phonation and deglutition which are generally first and always most constantly and extensively affected: the mucous membrane of the vocal processes, the inter-arytenoid region, the vocal cords, the cartilages of Santorini, and the aryteno-epiglottidean ligaments. Here are established both the more acute superficial losses of substance just mentioned, and the chronic ulcers which no doubt chiefly originate from the follicles.

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[1] **Pathol. Anatomie. Bd. II. p. 312.**

It is manifest that the latter will be most frequent in those localities in which the racemose glands are most abundantly situated, and which at the same time are liable to frequent and energetic mechanical irritants; such a locality is the posterior wall of the larynx, the inter-arytenoid region, the mucous membrane of which, at every phonation, is thrown into fine vertical folds, by the contraction of the arytænoideus muscles and the consequent juxtaposition of the arytenoid cartilages, these folds disappearing again as they separate. This folding of the mucous membrane during phonation amounts to a veritable pressure when the mucous membrane or glandular tissue is thickened by catarrh, and on the juxtaposition of the cartilages is crowded forward between the vocal processes as a fold (laryngoscopically visible). If there is now a specific catarrh, with abundant cell-infiltration of the mucous membrane, termination in a destructive process must certainly be materially favored by these mechanical injuries.

Follicular ulcers at first present losses of substance very little extended in surface, but penetrating all the more in depth, with margins thrown up like walls, and which may afterwards grow through confluence and cause considerable destruction. The mucous membrane of the vocal processes and the posterior wall of the larynx at the base of the arytenoid cartilages is destroyed, down to the perichondrium and to the cartilage itself; on the vocal cords the mucous membrane looks eaten out—indeed, complete destruction of the mucous membrane, advancing from behind forwards, with or without loosening of the cord from its process, is often seen. The changes which may be produced in the cartilages and articulations, by penetrating ulcers—the perichondritis, necrosis of the cartilage, disturbance of the crico-arytenoid articulation—have already been treated of above under the pathogenesis of perichondritis. The part which phthisical ulcers may play in the production of acute laryngeal œdema has also been mentioned when treating of phlegmonous laryngitis.

Ulceration of the mucous membrane on the posterior surface of the arytenoids and the plate of the cricoid very often takes place, through the acute and flat form of ulcers, towards the end of life; the infection, indeed, appears to creep from the inner surface of the arytenoid region, over the upper border of the arytænoideus muscle, backwards into the pharynx, just as it likewise extends to the aryteno-epiglottidean ligaments and the inner surface of the epiglottis. Thus the entire inner surface of the upper larynx may finally be ulcerated, and, through the participation of the muscles, the cartilages and the joints, as well as œdematous swelling of the submucous tissue, the functions of the larynx may be deeply injured.

It is undoubtedly possible for the chronic ulcers to terminate in *healing*—isolated instances of this kind are communicated by various authors, and I myself have treated two such cases in which the cicatrix could be demonstrated on post-mortem examination—but such a result is very rare. The cheesy degeneration having ceased, a permanent connective tissue may form at the base and in the neighborhood of the ulcer, which finally contracts to a radiated cicatrix.

It is true that new ulcers generally develop afterwards, so that the healing of a single one is after all of very little prognostic significance.

We have already, above, given prominence to the fact that the pathogeny of laryngeal phthisis presupposes, as an etiological basis, the existence of a hereditary or acquired disposition thereto. An important question which must here be discussed relates to *the order of time or priority existing between laryngeal phthisis and pulmonary phthisis.*

The view which passed current at the beginning of the nineteenth century, that laryngeal phthisis might, in rare cases, arise without previous pulmonary consumption, and might even itself produce the pulmonary affection, was overthrown by Louis with the assertion that pulmonary phthisis always preceded laryngeal phthisis, even though it might not always be demonstrable. This view of Louis has been extensively opposed. I mention among its opponents only Trousseau and Belloc, Albers, Andral, Ruehle, Waldenburg, Ter Maten, Sommerbrodt; but the opposition is, no doubt, only directed against the absolute negation.

Every clinician must surely recognize it as *the rule*, that laryngeal ulceration is preceded by a destructive pulmonary affection; and, according to my experience, I should designate it as a rule which has but few exceptions.

For about fifteen years I have given special attention to this subject, having a large amount of material for observation at my command, and thus far I have *not found a case* of phthisical ulcers of the larynx in which I could not demonstrate either fresh or old consolidations in the lungs, especially at their apices. The careful examination of the apices of the lungs, with reference to the air they contain, and especially with regard to their *comparative height*, which E. Seitz, of Giessen, first recommended, and the significance of which I first urged before the meeting of Naturalists and Physicians in Frankfort in 1867 (Section for Internal Medicine), is of cardinal importance in the decision of the very question before us. The flattening of one apex as compared to the other permits one to recognize slight cicatricial contractions of the parenchyma at a time when the patient, without regard to this point, must have been pronounced sound, as to his lungs, as even the signs of catarrh of the apex and of diminished air-contents may fail in individual cases.

I will not deny that exceptions to this rule occur, and that other observers have seen what I could not observe. As Buhl[1] has already shown, nothing wonderful is to be found, *à priori*, in such an appearance of laryngeal phthisis, as in a well-pronounced phthisical constitution the first eruption may take place in some other organ than just in the lungs, and sometimes does so take place, and as, furthermore, we cannot deny the possibility that pieces of detritus thrown off by the laryngeal ulcers and coarser pieces of tissue may be drawn into the lung during inspiration and here produce phthisis. But I would assert, on the ground of clinical experience, that such an order of development is certainly very rare indeed.

Sommerbrodt has sought directly to demonstrate the possibility of a secondary development of pulmonary disease from a primary laryngeal suppuration by experiments on animals. In squirrels who, so far as their constitution is concerned, seem to occupy the same place amongst animals that persons with a hereditary predisposition to phthisis do in the human race, he produced acute inflammation, caseation and suppuration by mechanical irritation of the larynx (drawing through it a fine iron wire, heated red), following which, in the course of some days or weeks, was a pulmonary disease which Sommerbrodt is disposed to regard as *peribronchitis purulenta*, although the purulent breaking down of the peribronchitic cell-infiltration was not observed, perhaps by reason of the too early death of the animals. In dogs mechanical interference did not have this result.

However interesting the result of these experiments is, they have, after all, no direct bearing on the question of the relative time at which laryngeal phthisis in the human subject is developed, as the primary disturbances here are essentially different.

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[1] In Sommerbrodt, l. c., p. 265.

As regards the *frequency of laryngeal phthisis*, according to the statistics of Willigk, which are generally cited, it is not as frequent, in comparison to pulmonary and intestinal phthisis, as is commonly supposed. But this question undoubtedly demands renewed statistical investigation, based on the largest possible amount of material. According to Willigk,[1] 1317 "tuberculous" bodies showed laryngeal affections only 237 times—that is, in 13.8 per cent. (14.3 per cent. of men, 11.3 per cent. of women); while intestinal affections were shown in 49.6 per cent. of all the cases.

Symptomatology.

The precursors of tubercular laryngeal affections are often very insignificant evidences of functional weakness of the organ: a certain sensitiveness of the organ, a lack of ring to, and ready failure of, the voice at every exertion (speaking, singing), the occurrence of hoarseness on taking a slight cold. Laryngoscopic examination shows partial injection and swelling of the vocal processes, of the inter-arytenoid region and the cartilages of Santorini; but often, also, quite the opposite—a striking anæmia of the mucous membrane and not seldom paresis of the muscles. These changes, in themselves, would not give occasion to any serious alarm, if the family history did not reveal phthisis in one parent, and careful examination of the apex of the lungs did not show changes of some kind (dulness on percussion, flattening of the apices, an expiratory murmur audible in the subclavian region (Ruehle), catarrhal sounds, prolonged expiration, weakness of the respiratory murmur.

With proper care and appropriate medical treatment this irritability and anæmia of the mucous membrane, as well as the muscular weakness, may entirely disappear; but in such patients, in spite of all care, the larynx remains a weak point. The catarrhal conditions finally become permanent. Erosions and flat ulcers, surrounded by a narrow injected areola, are now developed on the vocal process or between the arytenoids, seldom, just at first, in other parts of the larynx. The ulcers at the base of the arytenoids, as well as those between the cornicula, at first often show nothing but their jagged edges, which sometimes sprout up like papillæ. It is only when the plate of the cricoid and the arytenoids are naturally directed strongly backwards, and when the head is bent far back, that it is possible to obtain a view of such ulcers upon the inclined plane thus presented.

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[1] Prager Vierteljahrschrift, 1856, XIII. 2, p. 10.

The accompanying drawing of Türck's gives an instructive picture of this.

Catarrh of the surrounding soft parts seldom fails; the injection and swelling of the mucous membrane is developed to a varying degree; habitual exertion of the voice, and other injuries, of course have an influence essentially for the worse. The *disturbances of the voice*, the hoarseness, which may even advance to aphonia, the impurity of the voice, the easy exhaustibility of the power of phonation, depend in each individual case on the amount of catarrhal changes that accompany the ulcer and on any paresis or paralysis of the muscles that may exist.

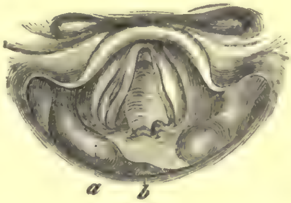

FIG. 11.

Phthisical ulcer on the posterior laryngeal wall. Secondary perichondritis laryngea.

With reference to the motor disturbances which occur here so often, we must distinguish between the muscular paresis which is frequent at the beginning of laryngeal disease, and which appears to depend on the general anæmia of the soft parts within the larynx (Rud. Meyer), and the paralysis of the right vocal cord, dependent upon paralysis of conduction of the right recurrent laryngeal nerve, which is often found imbedded and constricted in the hard, thickened pleural tissue about the apex of the right lung (Gerhardt). The former, as Rud. Meyer has demonstrated, may be quickly removed by appropriate treatment, while the paralysis of the recurrent laryngeal just described is inaccessible to treatment. Finally, the existence of muscular paresis in the final stages, when inflammatory œdema has infiltrated the submucous tissue and sarcolemma, may be explained by this circumstance, notwithstanding that it is hard to prove, as the intense œdema of the soft parts and the changes in the cartilages and the crico-arytenoid articulations at the same time cause mechanical hindrances to the locomotion of the arytenoid cartilages.

In cases in which there is no paralysis, in which the ulcers are small and hidden, and the catarrhal disturbances slight, every reason for suspecting the presence of an ulcer may be lacking, and its existence may only be established on post-mortem examination.

Cough, so far as it depends upon the laryngeal trouble alone, is generally very insignificant; it expels at first mucus, afterwards muco-purulent sputa, to which little shreds of blood are sometimes attached. According to Ruehle, in rare instances of deep ulceration, it is possible to demonstrate the presence of straight elastic fibres, which can easily be distinguished from the looped, characteristically arranged elastic elements of the lung tissue.

At first, and sometimes during the entire course of the affection, no complaint is made of *pain;* but in some patients, who complain of no spontaneous pain, this may be produced by pressure on the larynx, especially if the attempt is made to grasp it from both sides at once at the height of the arytenoid cartilages.

The manifestations of phthisical laryngeal ulceration appear much more intense when the superficial, more acute ulceration spreads itself over the greater part of the interior of the larynx and the trachea, over the inner surface of the epiglottis and the posterior surface of the arytenoids—as is the case in all instances in which the larynx is prominently affected, especially towards the end of life. In these cases there is generally a burning pain, which is increased on speaking, swallowing, and through the irritation of the abundant muco-purulent secretion; every act of swallowing becomes torture, and all the patient's complaints are concentrated upon this difficulty of swallowing. The almost constant œdematous infiltration of the submucous tissue of the epiglottis and the aryteno-epiglottidean folds prevents the complete closure of the laryngeal entrance, so that even on the most careful swallowing of fluid nutriment a small quantity of it flows into the larynx every time, and provokes the most distressing paroxyms of cough.

The laryngeal mirror permits us, to a great degree, to look over the extensive ulcerated surfaces, and by showing the circumscribed œdemas and the immobility of one or both arytenoids, or the defects in the epiglottis, enables us to form an opinion as to the secondary involvement of the perichondrium and destruction of the cartilages.

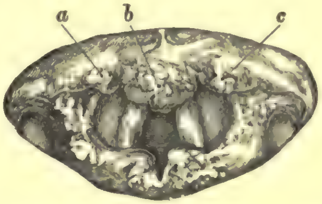

FIG. 12.
Extensive phthisical ulceration of the larynx. *a*, *b*, *c*, Remnants of the epiglottis. After Türck.

The *cough*, during this stage, is generally without tone or power, on account of insufficient closure of the glottis, and for this reason is very fatiguing to the patient. The voice is usually entirely lost. Ulcerative destruction of the vocal cords, their loosening from the vocal processes, immobility of the vocal cords and of the arytenoids as the result of necrosis of the cartilage, of muscular paralysis, or of dense œdema, are the usual causes of the terminal aphonia. Not seldom one hears the patients, who are only able, as a rule, to make themselves understood in a hoarse whisper, at times speak up, though not without an effort, in a deep, rough, and monotonous voice. These deep, impure tones are caused, as one can easily satisfy himself laryngoscopically, chiefly by vicarious vibrations of the false cords, which, during phonation, are approximated to the median line by the thyreo-arytænoideus externus muscle, and are thrown into coarse vibrations by the current of air. The cough, too, is accompanied by the deep sonorous vibrations, though it sounds flat and powerless, as the energy of the expiratory muscles as well as the power of closing the larynx is reduced to a minimum.

Diagnosis.

As a general rule, no difficulty is found in establishing the diagnosis of ulcerative laryngeal phthisis, when, in addition to the alterations above described, the symptoms of pulmonary phthisis can be clearly demonstrated. Not rarely, however, it becomes a very difficult matter, at first, if the diagnosis of a pulmonary affection is not established beyond all doubt, especially if a moderate but obstinate catarrh and a limited ulceration exists in a well-nourished person not hereditarily predisposed, or, when this state of things is found in a nervous subject, especially a woman, accompanied by muscular paresis. Here we must depend for a decision upon the further course of the affection, and especially upon the result of systematic local and general therapeutic interference. Neither the catarrh nor the ulceration of phthisical subjects presents any characteristic signs by which it could be recognized as such. The attempts made to establish pathognomonic peculiarities cannot be said to have succeeded.

Under some circumstances, the differential diagnosis between phthisical and syphilitic ulcers may present great difficulties. Generally we find a sufficient number of fixed points for our guidance in the evidences of syphilis in the pharynx, the fact of the descent of the ulcerations from the lateral portions of the pharynx on to the epiglottis and the aryteno-epiglottidean folds, the existence of great defects in the epiglottis, and, on the other hand, the absence of pulmonary phthisis, as well as the character of the ulcers themselves (see next chapter). But when, as is tolerably often the case, evidences of pulmonary disease, with simultaneous ulceration of the larynx, appear in a person suffering from old syphilitic trouble in the pharynx, we may, under some circumstances, in spite of the most careful weighing of all points of diagnostic importance, be forced to base our diagnosis more on the results of treatment (ex juvantibus et nocentibus).

According to my experience, it is hardly possible to confound tubercular ulcers with the rare lupous ulcerations of the epiglottis sometimes found. The same is true with regard to typhous ulcerations, whose origin, indeed, would render mistakes very unlikely.

Course and Prognosis.

The course of laryngeal phthisis is always chronic, but within the compass of this expression there are very considerable extremes of duration. As a general rule, one may say that in each individual case the course of the laryngeal affection is determined by the character of the pulmonary phthisis; the more acute the course of the pulmonary phthisis, the more rapid is the spread of the laryngeal ulcers, and vice versa. Patients may be found, in whom phthisis is progressing very slowly, who suffer for years from laryngeal ulcers without their spreading to any considerable extent; while there are others, in whom pulmonary phthisis is advancing pretty rapidly, where ulceration of the laryngeal mucous membrane runs almost an acute course and spreads over very large surfaces within a short time. In the first class, undoubtedly the destruction of the laryngeal mucous membrane may come to a standstill and even result in actual healing—that is, cicatrization of the ulcer. All authors agree about this. But these cases are, after all, very rare, and even such patients generally perish of pulmonary consumption at last, even though it may not be for years, with relapses of laryngeal phthisis. We have, therefore, every reason to make a uniformly unfavorable prognosis in cases of ulcerative laryngeal phthisis, and not to expect too much even from the most careful rest of the organ and the most judicious medical treatment.

The appearance of œdema of the glottis or of perichondritis of course renders the prognosis much worse, and the necessity for tracheotomy is usually the beginning of the end.

Treatment.

The treatment of laryngeal phthisis, as was remarked under the head of prognosis, offers, it is true, but little prospect of great and permanent results, but it is, nevertheless, by no means useless. For, aside from the few cases in which one succeeds in bringing a tuberculous ulcer to heal, one is almost always able to effect a material amelioration of the annoying symptoms.

In the way of *prophylaxis*, in case of suspicious laryngeal catarrh in persons with a hereditary predisposition to pulmonary phthisis, or with pulmonary affections already developed, the greatest care is requisite in avoiding all injurious influences (speaking, singing, smoking, or remaining in smoky or dusty air, or in air kept at too low or too high a temperature). In addition, the water of the Ems Springs should be used, mixed with milk or whey—best used, systematically, at Ems itself,—the application of mild astringents (weak solutions of nitrate of silver, tannin, or alum) is to be employed, either in the form of solutions applied with a brush, or of powders insufflated. If the mucous membrane is very irritable, pencilling the interior of the larynx, from time to time, with solutions of morphine and bromide of potassium, as well as with pure tincture of opium, is very highly to be recommended, so also the cold compresses of Priessnitz and derivation to the outer skin of the neck.

If *ulcers* already exist, the same general and local treatment is still, on the whole, to be adopted: perfect silence is to be maintained during weeks and months; care is to be taken to preserve a uniform temperature; during the unfavorable season of the year a residence at the Riviera (San Remo, Mentone) is to be recommended, or at least on Lake Geneva (Montreux), or in Southern Tyrol (Botzen, Gries, Meran); or, finally, if circumstances do not admit of a change of climate, Jeffrey's respirator is to be worn on walking out in winter, the waters of Ems are to be systematically used, with or without whey; and, finally, the not too frequent or too energetic application of astringents is to be employed. Touching with nitrate of silver in substance or in strong solution, which acts so favorably in simple chronic catarrh and in syphilitic ulcerations, must generally be abandoned here, as, by reason of the great vulnerability of the mucous membrane, infiltrated with cellular elements, this treatment is generally followed by the further extension of disintegration in the neighborhood of the ulcer and new infiltration. But in spite of this, as Schnitzler appropriately remarks, one is often obliged, at the request of the patient, when the pain and difficulty of breathing has become intolerable, to cauterize the entire ulcerated surface energetically with lunar caustic, for the sake of diminishing, at least for a short time, the sensibility of this surface.

Very favorable palliative results may be obtained from pencilling the interior of the larynx with tincture of opium, solutions of morphine, bromide of potassium, or bromide of ammonium, in water or in glycerine, from fifty to one hundred grains to the ounce (Gibb, Waldenburg). In very extensive ulcerated surfaces, with an abundant secretion, I can recommend pencilling with solutions of chlorate of potassa (twenty grains to the ounce).

Above all, one should not allow himself to be led away by the urgency of the patients, who, with undue confidence in local treatment, present themselves too frequently during his office hours, into the continuous application of astringents. One must confine himself, in the application of the aforenamed narcotics and antiseptics, to diminishing the irritable condition and the secretion of the ulcerated surfaces and their surroundings,—an end which will often be admirably accomplished, even though but for a short time.

Inhalations of the above-named solutions of bromide of potassium, chlorate of potassa, chloride of sodium, morphine, etc., atomized, and used for two or three minutes twice a day, are also admirable palliatives. The use of moist, warm compresses to the neck, as well as the application of counter-irritants of longer duration (croton-oil, blisters), may be of service in active catarrhal irritation of the entire mucous membrane of the larynx and trachea.

Lupus, Lepra, and Glanders of the Larynx.

LUPUS: *Türck*, Zeitschrift der Gesellschaft d. Aerzte zu Wien, 1859, No. 11; and Klinik, p. 425. Atlas, Taf. XX. 6, and XXI. 1.—*Waldenburg*, Lehrbuch der Respirator. Therapie. II. Aufl. 1872, p. 834.—*Virchow*, Die krankhaften Geschwülste. Bd. II. p. 490, ff.

LEPRA: *Daniellsen* und *Boeck*, Traité de la spedalskhed. Paris. 1848.—*Wolff*, Virchow's Archiv. Bd. XXVI. p. 44, ff. 1863.—*Virchow*, Die krankhaften Geschwülste. Bd. II. p. 519.—*Hillairet*, Mémoires de la Soc. de Biol. 1862. Sér. III. Tom. IV. p. 224, cited in Virchow, l. c.—*Gibb*, Diseases of the throat and windpipe. II. Edit. London. 1864.—*Schroetter*, Laryngol. Mittheilungen. II. 1874, p. 84.

GLANDERS: *Hauff*, Die Rotzkrankheit beim Menschen. Stuttgart. 1855.—*Virchow*, Die krankhaften Geschwülste. II. p. 552; and his Handb. d. spec. Pathol. u. Ther. Bd. II. p. 416.—*Korányi*, Zoonosen im Handb. d. allgem. und spec. Chirurgie. Bd. I. 2.—*Bollinger*, this Cyclopædia. Vol. III. p. 362.

Lupus.

Lupus of the larynx, to judge by the scant mention of it in literature, is a very rare affection. I cannot pretend to say whether more careful laryngoscopic studies, in regions where lupus is abundant—both of which conditions are essential—would yield different results. I can only form the conjecture, from the frequency with which I observed hoarseness and other laryngeal disturbances during pre-laryngoscopic times, in my first field of labor on the Pomeranian coast of the Baltic Sea—a region very rich in lupus, and the cradle of the valuable investigations of Pohl and Berger—that the larynx is oftener the seat of lupous disease than has hitherto been believed.

The *anatomical changes* that take place in lupus of the larynx are described by Virchow (l. c.). In one case an indurated cicatrix extended from the middle of the dorsum to deep down in the root of the tongue, while thick knobs, up to the size of a pea, rose alongside of it. The epiglottis was to the highest degree hard, and its edges raised in welts; a knotty hardness extended from there down into the trachea; the vocal processes of the arytenoid cartilages were the seat of deep ulcerations, which were surrounded by hard papillary outgrowths.

The lupus nodules consist (Virchow, Auspitz, Pohl) of a young, soft, generally very vascular granulation tissue, which contains little round cells, and originates in proliferation of the connective tissues, not of the epithelium. The termination of lupus, in the mucous membrane as well as in the skin, is in ulceration with progressive destruction. The healing of defects, with an indurated cicatrix, is seldom lasting; usually new miliary eruptions break forth in the immediate vicinity of the scar.

The first laryngoscopic examination of laryngeal lupus was effected by Türck, to whom we are indebted, indeed, for all the most valuable conclusions with regard to this affection. Türck examined four cases in all (to which, undoubtedly, a fifth one should be added), and established the following points: In the four unequivocal cases there were ulcers on the epiglottis, with loss of a part of the free edge throughout its entire thickness—

chiefly in the form of a heart-shaped piece cut out of the middle, as in syphilis or cancer ; in one case it was cut square across, as in syphilis. Aside from this, in three cases there were growths present, rather large elevations like fleshy warts, or smaller, irregularly jagged or simply round ones, on the anterior surface of the posterior wall of the larynx. On the soft parts of the pharynx there were similar granulation nodules and ulcers.

The accompanying illustration represents the condition of things as shown by the mirror in Türck's case during the year 1864 (l. c., p. 428). The patient was a man forty-five years of age, who had a primary chancre in 1848, was taken sick with lupus of the lip and nose in 1859, of which he had had a relapse since, but which had not appeared within the past three years. For a year past he had suffered from hoarseness and occasional dyspnœa. Very numerous reddish growths, of the size and shape of fleshy warts, though more elevated and consistent than the latter, were found over more than half of the hard and soft palate, over the palatine arches of the right side and over the entire anterior surface of the uvula. Below the palatine arches the lateral walls of the pharynx were partly ulcerated out, and the mucous glands at the base of the tongue were greatly swelled. The free border of the epiglottis shows a deep, triangular, ulcerative indentation, nearly splitting it in two. Over the entire upper division of the posterior laryngeal wall and on the aryteno-epiglottidean folds there are extremely numerous, larger and smaller, roundish growths, similar to those on the palate, and partly ulcerated. The vocal cords could not be seen. There was intense hoarseness, the cough being sharply limited. Swallowing and pressure on the larynx were painless. Compare also the pictures in Türck's Atlas, Plate XX. 6, and XXI. 1.

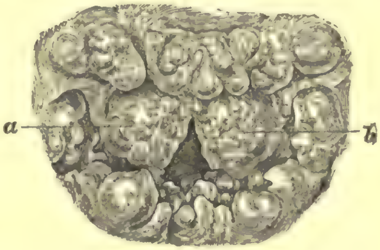

FIG. 13.
Lupus of the larynx. After Türck.
a, Right half;
b, Left half of the epiglottis.

A case observed by me corresponds entirely with Türck's examination, differing only in the circumstance that neither lupus of the skin nor of the pharynx appeared.

G. N., twelve years of age, the daughter of a Protestant minister, had for years suffered from hoarseness without any difficulty of breathing. She had thus far presented no other symptoms of disease, especially no sort of eruption, neither on the face nor on the body. The patient looked fresh and blooming. No exanthem on the face. The pharyngeal mucous membrane quite free. The epiglottis on the left side was quite defective; at the site of the loss of substance, and in the immediate vicinity, there were numerous granulation nodules, which spread to the aryteno-epiglottidean folds and the false cords of the left side. Syphilis was positively to be excluded, both from the previous history and from objective examination of the rest of the body. Furthermore, the administration of iodide of potassium for months, coupled with energetic local treatment, had produced no effect worth mentioning. Six months' treatment with cod-liver oil, and strong cauterizations of the new-growth with the stick of nitrate of silver, at least had the result of bringing the process to a stand-still.

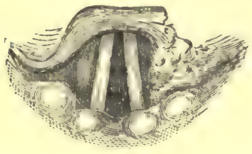

FIG. 14.
Lupus of the larynx.

The nature of this case was, at first, difficult to determine, owing to the absence of lupus of the skin, but its subsequent course, as well as the result of treatment, completely established the diagnosis.

The *course* of lupus in the larynx, as on the mucous membranes of the nose, pharynx, and external skin, is chronic. The question of whether the processes of new growth and destruction can spontaneously limit themselves, must be left to further observations to decide.

Prognosis and therapeutics.—The prognosis is, to say the least, doubtful. When we take into consideration the obstinacy of lupus new-formations, in general, over against the best of treatment, and their especial tendency to relapses, taken in connection with the dangers which arise from the seat of this destruction being at the entrance of the larynx, the prognosis might well be designated as very unfavorable.

The only guides that I have to estimate the value of therapeutic measures are, unfortunately, the one case of Türck's (Klinik, p. 427) and my own.

In Türck's case, which was that of a girl eleven years old, who had been troubled with lupus of the face for three years, and who for two or three years had suffered with hoarseness, frequent pain in the larynx, and occasional dyspnœa, especially at night, the upper division of the free border of the epiglottis was carried away through its entire width, as if by a transverse incision. The mucous membrane of the entire anterior surface of the upper division of the posterior laryngeal wall was degenerated in the form of great, round, transverse elevations, extending far down; further down smaller excrescences appeared, and these smaller ones were also found on the posterior surface of the epiglottis. Still smaller growths occupied the inner border of the swelled, uneven vocal cords. The voice was very hoarse, almost whispering; on attempting to phonate, the vocal cords remained moderately gaping.

After a year and a half, during which time the patient was in attendance on Hebra's division, and for a long time took cod-liver oil, the lupus of the face was materially better; the difficulty of breathing was diminished; the elevations on the anterior surface of the posterior laryngeal wall were somewhat flattened; the vocal cords were in about the same condition as before—their upper surface uneven, partly cicatricial and reddened in spots, the inner border of the left cord, throughout almost its entire length, having a glandular appearance.

As in this case of Türck's a retrograde development of the new-growth, at least to a great degree, could be proved, so also in my case, given above, an arrest of the process and partial shrivelling of the new-growth was observed under the treatment with large doses of cod-liver oil and strong cauterizations with nitrate of silver. Still, we could by no means on this account depend upon the permanent cessation of the process. There is urgent need of further observations, both with regard to laryngeal lupus generally, and in particular with regard to its therapeutics.

The *treatment*, based on the principles which hold good for lupus of the skin and of mucous membranes generally, must be confined especially to the long-continued use of cod-liver oil internally, together with salt-water baths, and, locally, to strong cauterizations with nitrate of silver in substance, destroying the new-growth.

Lepra.

Lepra (elephantiasis Græcorum) is accompanied by alterations in the larynx which, like the eruptions observed on the conjunctiva, then on the mucous membrane of the nose and the mouth, are histologically identical with the granulation tubercles of the external skin, and differ from them only in the disposition which the tubercles in this situation have to early ulceration.

The beginning of the changes within the larynx is always in the form of tubercular granulation, which, according to Virchow, at a certain period presents great similarity to syphilitic mucous papules or to follicular buboes, it being possible to distinguish them from these only by their greater hardness and vascularity. Sometimes the granulation-tissue does not appear in the form of tubercles, but as a grayish-white, non-ulcerating infiltration into the larynx (Hillairet[1]).

During the further course of events the tubercles ulcerate; the extent of the destruction in area and in depth seems to differ according to the severity of the case. In Norway, Virchow saw ulcerations of the larynx and trachea under which a hard, tendinous series of indurations was developed, extending deep through the submucous tissue and the elastic bands towards the outside, even the adipose tissue being involved in the leprous proliferation. Daniellsen and Boeck, too (l. c., p. 221), have described such destruction in the interior of the larynx. According to Virchow's investigations, the histological structure of the swollen mass is that of granulation-tissue. At first it consists only of simple spindle-shaped and stellate connective-tissue cells; by active cell-division and nucleus-division the inter-cellular substance becomes more and more scanty, so that only quite narrow bands of a feebly striped intermediate mass, which becomes cloudy and granular on the addition of acetic acid, remain to be seen between the rows and groups of cells. At the height of their development the cells represent round, pale, feebly granular, easily destructible elements, usually with a moderately large and likewise granular nucleus and a nucleolus. Their size varies between that of a red blood corpuscle and that of the largest mucus corpuscles. Virchow calls attention to the great similarity between this development and that of lupus; and, indeed, the entire process, as to its seat, its long duration, and its usually slow development, resembles lupus more than it does syphilis. It corresponds with both of them in the fact that the tubercles may after a time soften and ulcerate; but in general it is distinguished from them by its lasting much longer, its remaining nearly stationary under favorable circumstances for years—a quality, however, which, as above stated, is more peculiar to the affection when situated on the external skin than when on the mucous membrane, especially that of the respiratory tract. At the same time the tubercles do seem in many cases to escape ulceration till the end of life, as was shown in a case recently examined post-mortem by Thomas,[1] in which, besides pretty active hyperæmia and general swelling, the laryngeal mucous membrane was found sprinkled with numerous yellowish papules or tubercles, of the average size of a millet-seed. The same thing is shown in the investigations of Schroetter.

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[1] Cited by Virchow, l. c.

With regard to *etiology*, we must refer our readers to the more detailed discussion of this subject under the general head of Skin Diseases, in a later volume of this Cyclopædia.

Concerning the *symptoms* of laryngeal lepra, Virchow's studies have shown that even during the Middle Ages hoarseness and dyspnœa played an important part in the inspection of lepers, inasmuch as the "vox rauca" passed as an unequivocal sign of leprosy.[2] In very recent times the symptoms on the part of the larynx have been more accurately studied, with the aid of the laryngeal mirror, by C. Wolff, Gibb, and Schroetter. Wolff had the opportunity on the island of Madeira of making laryngoscopic examinations of seven cases of leprous laryngeal affection. In all of them he found chronic catarrh of the larynx; the epiglottis swelled, reddened, and highly vascular; the same condition of the aryteno-epiglottidean folds; the mucous membrane of the arytenoids and the false cords loosened, much thickened, colored dark-red or even bluish-red, and secreting copiously; the vocal cords, in the further course of the affection, generally strongly injected and thickened, and yellowish-red. Besides these changes, which partake of the character of intense chronic laryngitis, he always found growths on the mucous membrane, varying from the size of a pin-head to that of a pea, of a bright-red color and distinctly recognizable papillary structure, situated on the epiglottis, the arytenoid cartilages, and the false cords—seldom on the true vocal cords. Disturbances of motion come into view with the progress of the ulceration. The voice is very hoarse and without ring.

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[1] Beiträge zur patholog. Anatomie der Lepra Arabum. Virchow's Archiv. Bd. 57, p. 455, 1873.

[2] "Das erst zeychen ist die heyssere in der stymm vnd red, enge des otems." *Hans von Gersdorf*, Feldbuch der Wundartzeney, 1526, Bl. LXXXIV.; quoted in Virchow, l. c., p. 519.

In a case of advanced laryngeal lepra, Gibb found (l. c., p. 272) extensive defects of the epiglottis and the vocal cords, with considerable thickening of the remaining parts of them as well as of the false cords and the aryteno-epiglottidean folds.

Schroetter, who made laryngoscopic examinations of the Norwegian "Spedalskhen" in the hospitals of Hernösand, Drontheim, and Bergen, found in lighter cases uniform thickening of the soft parts or isolated tubercles, sometimes at one point, sometimes at another. The severer cases showed narrowing of the laryngeal entrance, through rolling inwards of the lateral borders of the epiglottis, and swelling of the aryteno-epiglottidean folds; or closure of the entire laryngeal cavity down to the calibre of a lead-pencil; or, finally, extensive ulceration of the new-growth. Narrowing of the calibre of the larynx of course brings with it dyspnœa and suffocative attacks.

The *prognosis* of laryngeal lepra, like that of the disease in general, is highly unfavorable. Up to the present time nothing is known of *therapeutic attempts* to reach the laryngeal affection.

Glanders.

Glanders of the larynx, according to Bollinger, is much more frequent, in the acute as well as in the chronic form of glanders of horses, than was formerly believed. Bollinger estimates the frequency of laryngeal glanders at from fifty to sixty per cent. of all cases of the disease. Amongst men Hauff found the larynx affected eleven times out of thirty-five cases (thirty-one per cent.). The changes consist in the development of the yellowish nodules or tubercles and ulcers of glanders, which are situated upon a highly inflamed mucous membrane, and may by degrees occasion serious destruction, proliferation, and œdematous swelling of the mucous membrane.

The neoplasm of glanders is almost always also found simultaneously on the mucous membrane of the pharynx, the trachea, and the bronchi.

The *symptoms* consist, in cases of moderate infiltration of the laryngeal mucous membrane, in hoarseness, and a disposition to cough, with expectoration of a tough, and subsequently bloody, muco-purulent secretion. If the infiltration extends seriously, the secondary œdematous swelling of the entire mucous membrane gives rise to the symptoms of laryngeal stenosis,—of laryngeal œdema. This is accompanied by fever, very rapid pulse, and emaciation. All observers unite in testifying to the similarity of the picture of disease presented by chronic glanders in the human subject, especially when localized in the respiratory tract, and tuberculosis.

No laryngoscopic examinations appear as yet to have been instituted in this disease.

When the disease terminates in recovery, severe and lasting disturbances of the functions of the larynx may result, even after the complete healing of the ulcers, through the gradual shrivelling and contraction of the cicatrices. Bollinger (l. c.) refers to such a case, in which, as the result of cicatricial contractions in the nose and larynx, the patient suffered perpetually with cough and difficulty of breathing, and eleven years later presented the picture of pronounced cachexia.

Death, as the result of the laryngeal trouble, probably occurs only when œdema of the larynx takes place. No further particulars are known.

The average duration of the disease in subacute and chronic glanders is given by Bollinger as being about four months. About one-half of these escape with their lives, while the acute form almost always terminates fatally.

The *diagnosis* of laryngeal glanders, as of glanders generally in the human subject, is at first often exceedingly difficult—in fact, not seldom impossible. It is not until the occurrence of localizations on the skin, in the form of pustules, vesicles, abscesses, erysipelas, and phlegmon, or on the mucous membrane of the nose, pharynx, and larynx, associated with rheumatoid pains, fever, and the establishment of the general course of the affection, that we can arrive at a diagnosis, being then also aided by considering the occupation of the patient (as ninety per cent. of the cases of glanders are found in grooms, drivers, farmers, veterinary surgeons, etc.).

The *treatment* of glanders of the larynx, besides the general treatment of the disease, is confined to pencilling the pharynx and larynx with antiseptic fluids (solutions of carbolic or salicylic acid), together with the use of the same fluids as gargles, the inhalation of solutions of bromide of potassium with morphine, etc. On the occurrence of œdema, tracheotomy would be indicated.

Syphilis of the Larynx.

Morgagni, Epist. XV. 13. XXII. 26. XXVIII. 12. XLIV. 3.—*Thomann*, Annales instituti med. clin. Wirceburg. 1800–1803.—*Sachse*, Kehlkopf- und Luftröhrenschwindsuchten. Hannover, 1821.—*Albers*, Pathologie und Therapie der Kehlkopfkrankheiten. 1829, p. 182.—*Oppolzer*, Prager Vierteljahrschr. I. 1. 1844.—*Rheiner*, Virchow's Archiv. Bd. V. p. 577. 1833.—*Günther*, Prager Vierteljahrschrift. 1857. IV. p. 99.—*Pitha*, ibidem.—*Virchow*, Ueber die Natur der constitutionell-syphilitischen Affectionen. His Archiv Bd. XV. p. 307. 1858.—*Störck*, Zeitschrift der k. k. Ges. d. Wiener Aerzte. 1858. No. 51.—*Virchow*, Deutsche Klinik. 1860, p. 873.—*Gerhardt* und *Roth*, Ueber breite Condylome im Kehlkopf. Virchow's Archiv. Bd. XX. p. 402. 1861.—*The same*, Ueber syphilitische Krankheiten des Kehlkopfes. Ebendas. Bd. XXI. p. 7, ff. 1861.—*Ruehle*, Die Kehlkopfkrankheiten. 1861, p. 272, ff.—*Gilewsky*, Wiener medic. Wochenschrift. No. 18. 1861.—*E. Wagner*, Das Syphilom. Archiv der Heilkunde. Bd. IV. p. 221. 1863.—*Virchow*, Die krankhaften Geschwülste. Bd. II. 2, p. 412. 1865.—*M. Cohn*, Die Syphilis der Schleimhaut der Mund-, Nasen-, Rachen-, und Kehlkopfhöhle. 1866.—*Türck*, Allg. Wiener med. Zeitung. 1863. No. 43. Klinik der Kehlkopfkrankheiten. 1866, p. 377, ff.—Lehrbuch der Syphilis von *Zeissl.* II. Aufl. p. 205. 1872.—*Schnitzler*, Ueber Kehlkopfgeschwüre. Wiener med. Presse. 1868. No. 14, ff.—*Dance*, Éruptions du larynx dans la période sécondaire de la syphilide. Thèse. Paris. 1869.—*Morgan*, Syphilitic affections of the larynx. Med. Press and Circ. 1868. Octob. 14, 1869. Jan. 13.—*Sommerbrodt*, Die ulcerösen Processe der Kehlkopfschleimhaut in Folge von Syphilis. Wiener med. Presse. 1870. No. 20, ff.—*Fiedler*, Laryngo-Pharyngo-Stenosis syphilitica. Archiv der Heilkunde. 1871. III. p. 212.—*Elsberg*, Syphilitic membranoid occlusion of the rima glottidis. New York. 1874.—*Norton, A. T.*, Closure of the glottis by a syphilitic gummatous tumor. Transactions of the Patholog. Society. XXV. p. 38. 1874.—*Robinson*, A case of intra-laryngeal tumor in a syphilitic subject. Amer. Journ. Med. Sciences. April, 1875.—*Bucquoy*, On tubercular and syphilitic laryngitis. Gazette des Hôpitaux. April 20–29, 1875.—*Krishaber* and *Mauriac*, The laryngopathies during the first stages of syphilis. Annales de l'Oreille et du Larynx, Nos. 2 and 3. 1875.—*Poyet*, A contribution to the study of laryngeal syphilis. Annales de Derm. et de Syph. No. 4. Tome sixième, 1875.—*Péronne* and *Isambert*, A case of laryngeal syphilis, with total loss of the epiglottis, etc. Annales des Maladies de l'Oreille et du Larynx. Nov. 1, 1875.—*Isambert*, On pharyngo-laryngeal syphilis. Le Progrès Médical. Oct. 9, 1875. — *Maunoir*, A case of syphilitic laryngitis; tracheotomy, gummy tumors of the lungs. Bulletin de la Société de Paris. 1875. 269.— *Wagner*, On syphilis of the nose and larynx. Ohio Medical and Surgical Journal. June, 1876.—*Mauriac*, A lecture on some of the more severe syphilitic affections of the larynx, complicated by a peri-laryngeal phlegmonous inflammation. Annales de l'Oreille et du Larynx. No. 3. Tome I.—*Masson*, On the causes of asphyxia in syphilitic laryngitis, and its treatment. Thèse de Paris. 1875.—*Quioc*, A case of tracheotomy, made in extremis for asphyxia, due to a syphilitic laryngitis. Lyon Medical. No. 26. 1876.—*Bleynie*, On a case of syphilitic laryngitis; tracheotomy; cure. Journal de la Société de Méd. de la Haute-Vienne.

Compare also the general works of *Foerster* (Path. Anat. II. Aufl. II. p. 327), *Rokitansky* (III. Aufl. III. p. 23), *Albers* (Atlas d. path. Anat. II. Taf. 5. Fig. 6. u. 7), *Hope* (Morbid anatomy, Fig. 49), *Baeumler* (Syphilis, this Cyclopædia, Vol. III. p. 208), *Tobold* (Lehrbuch, III. Aufl. p. 283).

Etiology.

Syphilitic diseases of the larynx are not rare. It is true that no utilizable statistics with regard to the frequency of laryngeal syphilis as yet exist. For if a comparison is to be made between the frequency of laryngeal syphilis and the syphilitic affections of other organs, statistics must be based not only on the result of post-mortem examinations, but also (on account of the curability of the early forms of disease) on clinical and especially laryngoscopic investigations. In the meantime, we may yet deduce this much from the statements of authors: that laryngeal syphilis is more frequent than is generally believed.

According to Willigk's[1] reports of post-mortems in the pathologico-anatomical institute in Prague, which (during five years) covered 218 cases, the relative percentage of frequency with which the different organs were diseased was as follows: larynx, 15.1 per cent. (in six cases with a high degree of stenosis); bones, 56.4 per cent.; general cutaneous surface, 18.3 per cent.; mucous membrane of the palate, 17.9 per cent.; of the pharynx, 10.1 per cent.; of the nose, 2.8 per cent.; the lungs and air-passages, 2.3 per cent.; the rectum, 3.7 per cent.

Gerhardt and Roth, among 56 syphilitic patients, found, on laryngoscopic examination, that 18—or nearly one-third—had diseases of the larynx.

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[1] Prager Vierteljahrschrift. XXIII. 2. 1856, p. 20.

Engelsted[1] saw affections of the larynx 25 times among 521 cases of syphilis, that is, in 4.8 per cent. (among these 25 cases the epiglottis was affected 20 times).

Lewin,[2] among one thousand patients, found 44 (4.4 per cent.) in whom one could determine the presence of a laryngeal affection associated with hoarseness.

Sommerbrodt, too, according to his laryngoscopic observations, regards the involvement of the larynx as frequent. Among 84 syphilitic subjects he found ulcerative processes 15 times, and in 14 other patients catarrh and hypertrophy of the laryngeal mucous membrane; but these figures will not do to base statistics on, as the patients sought him on account of throat troubles.

As regards the *age* of those attacked, the rule which, from the nature of things, gives us a majority of all cases of syphilis during the years of youth and manhood, also applies to syphilitic disease of the larynx.

Gerhardt and Roth's 18 cases divide themselves as follows, with regard to age: in the second decennium there were 2; in the third, 9; in the fourth, 6; in the fifth, 1.

The influence of the *duration* of syphilis on the *frequency* of the laryngeal affection may be arrived at with tolerable certainty from the foregoing material, and is to the effect that the probability of a syphilitic invasion of the larynx, in any given individual, grows with the duration of syphilis generally.

Gerhardt and Roth found among 44 syphilitic subjects, with the early forms (secondary syphilis, according to Ricord), 11—that is, one-fourth—with laryngeal disease; while among 12, with the late forms (tertiary syphilis of Ricord), 7—that is, over one-half—were thus affected.

Sommerbrodt established the existence of laryngeal ulcers 10 times in 77 subjects of secondary syphilis (13 per cent.); while amongst 5 with tertiary syphilis, 3 (60 per cent.) had ulcerative laryngeal syphilis.

Concerning the *point of time* at which laryngeal syphilis is apt to appear, Sommerbrodt has tried, with regard to his own cases and those of Lewin and Türck, to establish the minimum period of time that has elapsed between infection and the outbreak of laryngeal syphilis. In Lewin's cases it was from two and a half to three months; in Türck's, six months; in Sommerbrodt's, five months. The latest period of the eruption of laryngeal syphilis Sommerbrodt found to be from five to fifteen years; Türck, as much as thirty years after.

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[1] Bericht über die 4. Abtheilung des Commune-Hospitals in Kopenhagen. 1867. Bibl. for Laeger. 5 R. XVII. p. 215. Cited in Virchow and Hirsch's Jahresbericht. 1868. II. Bd. p. 585.

[2] Die Behandlung der Syphilis mit subcut. Sublimat-Injectionen. Berlin, 1869.

Special interest is attached to an observation of laryngeal syphilis in a *nursling*, by Isidor Frankl,[1] as well on account of the rarity of the occurrence itself as for the opportunity of accurately following, in point of time, the course of the laryngeal difficulty.

The child was born on the 17th of April, and on the 24th of April was admitted to the Foundling Institution, well nourished (weighing six pounds) and perfectly well. On the 26th of June it showed coryza and syphilitic ulcers on the nates and labia majora (calomel was given). Nine days later there was bronchial catarrh; on the 12th day, hoarseness; from the 13th day on, manifestations of laryngeal stenosis appeared, which reached the highest grade on the morning of the 19th day, and caused death at noon.

Post-mortem examination showed, besides hypostatic pneumonia and syphilis of the liver, perichondritis of the plate of the cricoid cartilage, terminating in partial necrosis of the same, perichondritic destruction and loosening of the left arytenoid cartilage, a breaking through into the cavity of the larynx, indurated and fatty degeneration of the posterior crico-arytenoid muscles, the arytænoideus muscle, as well as the superior laryngeal nerve of the left side.

That the perichondritis here was syphilitic, Frankl thinks may be concluded with certainty in view of the clinical career and of the intense syphilis of the liver found on post-mortem examination.

The rapid course of the disease is furthermore highly noteworthy. About ten weeks after birth, syphilis first appeared; within ten days later death ensued through laryngeal perichondritis, and the autopsy showed extensive necrosis of cartilage, with breaking through of the abscess into the cavity of the larynx.

With regard to the beginning of laryngeal syphilis, most authors tell us that it not seldom arises when all symptoms of syphilis have long since disappeared, perhaps for years already, and the disease has long been regarded as completely extinguished. It often then appears combined with syphilis of the pharynx, but sometimes without any disease of the mucous membrane of the upper or lower pharynx or mouth—indeed, without any other syphilitic affection.

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[1] Ulcera syphilitica et Stenosis laryngis. Wiener med. Wochenschrift. 1868. Nos. 69 and 70.

The immediate occasion of the outbreak of syphilis in the larynx is universally and justly explained by the mechanical irritations and the atmospheric injuries to which the larynx is exposed in some occupations. We may say, quite generally, that those syphilitic patients who, from occupation or habit, subject the vocal organs to a great strain, or are exposed to frequent taking cold or other injuries, are much more disposed, *ceteris paribus*, to syphilitic disease of the larynx than other syphilitic subjects in whom this is not the case.

Of two patients in whom I observed a very late relapse of syphilis, confined to the larynx, one was an official at a custom-house on the frontier, who was obliged, by night duty, to turn out year after year, in all weather, and was seldom free from catarrh; and the other was a teacher in the public schools.

Some authors consider insufficient mercurial treatment of syphilis as a cause for the outbreak of the disease in the larynx.

Pathology.

Pathological Anatomy.

The syphilitic changes in the larynx appear under the form of simple catarrh, then of (broad) condylomata, of follicular hyperplasiæ, of gummata, of ulceration, terminating in defects of organs and the formation of large deforming cicatrices, of perichondritis with secondary necrosis of cartilage, finally of secondary papillary hypertrophy.

Syphilitic catarrh presents nothing characteristic. The reverse may be asserted of the simultaneous hyperplastic swelling of pre-existing lymphatic follicular structures, as of the tonsils, the root of the tongue, the posterior pharyngeal wall, which structures, according to Virchow, early assume a somewhat hard, gray, or whitish appearance, through cell proliferation; and, if this proliferation is copiously continued, may break down and ulcerate, leading to the formation of Virchow's follicular buboes, which this investigator compares to the buboes of the external lymphatic glands.

Condylomata, or *mucous papules*, which, according to Virchow, in spite of their resemblance to follicular hyperplasiæ, are quite different from them, proceed from the surface of the mucous membrane, and may, by taking on more of a gummy character, and breaking down like true gummata, become very much like follicular hyperplasiæ.

Gummy tumors are developed on the epiglottis and within the interior of the larynx, either singly or as a conglomerate nodular mass. According to Virchow, all stages of development may be traced in them. "Their beginning consists of little roundish elevations, similar to the follicles of the root of the tongue, but of a softer, more marrow-like consistency, often richly supplied with blood-vessels, and especially surrounded by varicose veins. These nodules ulcerate on their surface, at first form shallow ulcers, the surface breaking down and being cast off, and gradually penetrate in depth as new portions of tissue are involved in the change. The gummy tumor in this position, therefore, has neither the glutinous nor the firm character which it possesses in the periosteum or the external skin, but has a well-marked medullary, whitish or yellowish appearance, caused by the abundant proliferation of cell elements, which sometimes form the greater part of the nodules. If the ulceration reaches a certain depth, a purulent *perichondritis* usually becomes associated with it, which is combined with partial necrosis of the cartilage, and finally effects the expulsion of the necrotic pieces of cartilage, with the formation of deep, sinuous cavities. It is these deep laryngeal ulcers that bring about the much-dreaded stenosis, when they undergo partial or complete cicatrization" (Virchow[1]).

This clear description of the pathological processes in the syphilitic larynx, with which Virchow has lighted up this hitherto dark region, is completed by him through the clinical study of the conditions which follow, most important among which, next to the cicatrices producing stenosis, are destruction of the epiglottis, defects of the vocal cords, œdema of the larynx, and papillary hyperplasia.

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[1] Die krankhaften Geschwülste. II. 2, p. 413.

So far as cicatrization is concerned, Virchow considers the characteristic of the syphilitic cicatrix generally to be the unproductiveness of the actual cicatrization process and the contrast between this lack of productiveness at the centre and the excessive activity of growth at the periphery. He calls attention to the great similarity between these and the cicatrices of burns, with their unproductiveness and their subsequent severe contractions.

Perhaps there is no point in the body where the significance of these cicatricial deformities is greater than in the pharynx and larynx, certainly none where they interfere more materially with the functions of deglutition and respiration, so essential to life, or the important function of phonation. The form and character of the results of destruction and cicatricial contraction are very various. On the coast of the Baltic Sea I had the opportunity of observing and examining a large amount of material with reference to the results of syphilitic destruction in the larynx and pharynx. I saw the most manifold narrowings and distortions of the pharynx, cicatricial projections similar to a diaphragm, leaving only a narrow slit for swallowing and breathing ; defects of the epiglottis, in all forms and grades, up to the complete destruction of the same, or the enclosure of the remaining little stump in a circular cicatricial mass narrowing the upper laryngeal aperture; defects of one or the other arytenoid cartilage, of one vocal cord, etc.

Literature furnishes us with a large number of pertinent single observations. I only call attention, amongst those of more recent (laryngoscopic) times, to the observations of Türck (l. c.), Cameron and Gee,[1] von Fiedler (l. c.), Elsberg (l. c.), and Schech.[2]

The papillary hyperplasias, conical in form or picket-shaped, which are described as existing around syphilitic cicatrices (Virchow, Rheiner, Ruehle, Türck), may attain to a certain independence and add to the capacity of the cicatrix for encroaching on space.

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[1] Brit. Med. Jour. Dec. 21, 1867.

[2] D. Archiv f. klin. Medic. Bd. XVII. p. 259, 1876.

The disturbance of function reaches its climax through œdema of the laryngeal entrance, which is usually the product of perichondritis.

Symptoms, Diagnosis, and Course.

Among the symptoms of laryngeal syphilis, *hoarseness* is usually the first. The "raucedo syphilitica" has nothing characteristic, and may be produced as well by simple syphilitic catarrh or the development of condylomata as by syphilomata or ulcers. According to the seat and extent of the process, the hoarseness may be slight or may increase to aphonia.

Cough and *difficulty of swallowing* may be very moderate, or even entirely fail in early laryngeal syphilis. The same is true of *pain* which generally arises through pressure from without or during the act of swallowing, only when the rapidly advancing destruction has brought about an inflammatory œdema of the submucous tissue or a perichondritis. The syphilitic changes, in a stricter sense, generally run their course without pain (Ricord, Türck, Sommerbrodt, Tobold), a fact which, under some circumstances, may be of use in making a differential diagnosis; for instance, for the purpose of distinguishing them from tuberculous ulcers.

Difficulty of swallowing does not generally become serious until considerable defects in the epiglottis have supervened, or there is an eating out of the soft parts about the upper laryngeal aperture. The act of swallowing may, however, be performed normally—that is, without the entrance of food into the larynx—even when the entire supra-hyoideal portion of the epiglottis is wanting, and there is cicatricial deformity of the soft parts about the upper larynx, provided the patient is accustomed to this condition of things and swallows carefully.

This act is materially aided by the base of the tongue, which is automatically drawn back over the laryngeal entrance by the stylo-glossus muscle, and protects the glottis like a roof in the place of the epiglottis. This position of the root of the tongue, far to the rear, is, according to my observation, quite characteristic of defects of the epiglottis, and it renders laryngoscopic examination of such patients much more difficult. Evidently, however, closure of the upper laryngeal aperture is also aided by the contraction of the arytæno-epiglottideus muscle (of Santorini)—normally incomplete but hypertrophied by use,—the constrictor vestibuli laryngis of Luschka, which crowds the aryteno-epiglottidean folds and the arytenoid cartilages together towards the median line, transforming the upper laryngeal opening into a narrow antero-posterior slit. The juxtaposition of the false vocal cords, rendered possible through the action of the superior thyreo-arytænoidei muscles, also contributes towards preventing the entrance of food into the glottis.

The expectoration of muco-pus, mingled with blood, is a matter of course in case of large ulcers.

Through *laryngoscopic examination*, the kind, seat, and extent of the syphilitic processes are to be determined—indeed, first of all, the question of the syphilitic nature of the laryngeal affection is to be settled.

Syphilitic catarrh presents nothing more characteristic to inspection than to anatomical examination. It seems to me a very bold undertaking to conclude on the syphilitic nature of the process on account of the livid red, or dirty, brownish-red color of the injection of the mucous membrane, as these changes in the color of the injection may be developed in chronic laryngeal catarrh of all kinds.

The circumstance that simple, non-syphilitic laryngeal catarrh, with its changes for the better and the worse, may continue, obstinately, for a long time, and may gradually impart a dirty, reddish color to the mucous membrane, renders it impossible to pronounce on the syphilitic nature of catarrh from the color. The unusually prolonged duration of the catarrh, so valuable a sign in the diagnosis of the syphilitic nature of catarrh of the pharynx, loses all significance in the larynx.

One must, as a matter of course, take into account all other circumstances bearing on the diagnosis, especially the history of the patient, the examination of the genitals, of the pharynx, the skin, the lymphatic glands, the bones, etc. Finally, conclusions drawn from the effects of treatment are permissible, indeed, quite valuable in difficult cases, as general and local antisyphilitic treatment removes the catarrh surprisingly fast, which cannot be said of the treatment of tuberculous catarrh nor for that of simple subacute or chronic laryngeal catarrh.

The *broad condyloma*, arising simultaneously with the catarrh, or without it, appears as a flat, wart-like papule, with a thick, whitish gray, adventitious covering of epithelium, such as condylomata of the mouth and pharynx also bear, and asserts itself, from the outset, as a condyloma on account of its resemblance to condylomata generally (Gerhardt and Roth). Its seat is on the vocal cords, on the posterior laryngeal wall, and on the false cords. By reason of its seat, as well as the catarrh that exists around it, it interferes quite seriously, as a rule, with the formation of the voice. This serious interference with the voice was emphasized by Ricord[1] already, while Gerhardt and Roth first demonstrated, laryngoscopically, the conditions on which it depends. Mucous papules appear early (about six to ten weeks after infection), and generally at the same time with roseola and condylomata at other points of predilection (the fauces, genitals, anus).

At the same time it must here be stated, that on the part of some authors (Waldenburg,[2] Lewin[3]) grave doubts are raised as to the occurrence of mucous papules within the larynx.

The *gummy tumor* or *syphiloma* of the laryngeal mucous membrane, as such, has not yet been sufficiently studied on the living subject. The conditions and laryngoscopic pictures which Türck gives of subacute and chronic inflammation of the mucous membrane and submucosa, in syphilitic subjects, may, to a great degree, be considered as belonging here. Türck (Klinik, p. 380) describes the changes "on the true and false vocal cords as weals, running longitudinally; on the anterior surface of the posterior laryngeal wall, as rows of little hillocks, lying alongside of and under one another; on the mucous membrane covering the cartilages of Wrisberg and Santorini, as more or less roundish swellings, which often also occupy the cicatrized remnants of the epiglottis, whose jagged edges then appear raised to roundish or elongated roundish weals." Furthermore, according to Türck, these swellings, which have the color of the normal mucous membrane, may, when seated on the true or false vocal cords, lead to stenosis of the larynx, through participation of the submucous tissue, or may ulcerate out, in whole or in part, but can be made to recede by means of antisyphilitic treatment. The way in which the conditions found by Türck correspond with the alterations which Virchow describes as belonging to laryngeal gummata, scarcely leaves a doubt that in these cases the former was dealing, not with inflammatory products, but with specific syphilitic new-formations.

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[1] "If papules are developed in the larynx, the patient's voice will gradually go through with all the variations, from the slightest grade of dysphonia to the most complete aphonia. * * * * There is generally no pain. *Ricord's* Lectures, by Lippert, 1846, p. 80.

[2] Respiratorische Therapie II. Aufl. 1872, p. 366.

[3] *Lewin*, Die Behandlung der Syphilis mit subcutanen Sublimat-Injectionen, 1869, p. 113.

Waldenburg,[1] too, considers the smooth, generally roundish elevations, of the size of a pin-head to that of a small pea, usually of the color of the rest of the mucous membrane, found with special frequency on the posterior laryngeal wall, generally several of them together, and which he has often observed in the later stages of syphilis, as being gummata. These tumors did not commonly terminate in ulceration ; in fact, Waldenburg more frequently witnessed their absorption or obstinate persistence, even when all other symptoms of syphilis had already disappeared. Mandl[2] observed numerous gummata of a grayish-yellow color on the epiglottis and the false vocal cords of a negro, who also showed extensive syphilis of the pharynx. Of the handsome colored pictures which Mandl (l. c., Pl. VI. 2, and VII. 1.) gives of gummata, the last may here be reproduced in woodcut.

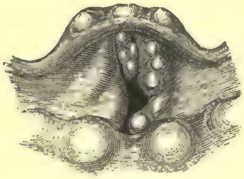
FIG 15.
Gummata laryngis. (After Mandl.)

A gummy tumor below the cords has been observed by Türck (Klinik, p. 389), Nicolas Duranty (Laryngite chronique ; Marseilles, 1865), and Norton (l. c.).

Ulceration of the gummy growth, syphilitic ulceration, appears in general to occur very early ; the fact that, in well-pronounced laryngeal syphilis, ulcers are found vastly oftener than neoplastic infiltrations, argues decidedly in favor of an early ulceration, caused partly, no doubt, by the mechanical irritations to which infiltrations are exposed on the epiglottis, the true and false vocal cords, and the posterior laryngeal wall.

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[1] *Lewin*, l. c., p. 113.

[2] Maladies du larynx. 1872, p. 700.

The depth of the ulceration, the participation of the submucous or muscular layer and of the perichondrium in the neoplasm and destruction, depends upon whether syphilis appears in the larynx in the form of an early or late affection ; whether, therefore, we have to deal with flat formations of the mucous membrane (condylomata and follicular buboes), or with gummata, penetrating deeply, even to the periosteum.

The more superficial affections may be healed, in spite of ulceration, through timely and appropriate antisyphilitic treatment, without leaving any defects worth mentioning in those parts of the organ attacked ; whereas ulceration of the deeply penetrating syphilomata results in those extensive destructions and those extremely deforming cicatricial masses which were described above (Pathological Anatomy).

According to my experience, *the extension of syphilitic neoplasms to the larynx by continuity* plays no unimportant rôle, and this circumstance is often of great value for diagnosis. In most instances, as is well known, destructive syphilis of the pharynx precedes that of the larynx. In appropriate cases, that is, in those coming under observation early, we can recognize the pharyngo-epiglottic ligament as the pathway by which syphilis travels from the pharynx to the larynx. For this reason, too, it is generally the margins of the epiglottis, on one side or the other, which are first infiltrated and soon also destroyed. From the margin of the epiglottis the neoplasm and destruction advance to the aryteno-epiglottidean ligament and to the inner surface of the epiglottis, or to both at the same time ; from both these regions the attack may extend to the false and the true vocal cords.

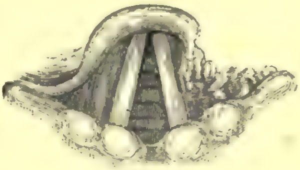

FIG. 16.
Syphilitic ulceration of the larynx.

The diagnosis of the syphilitic ulcer may be rendered much easier by this local behavior of the affection, especially by the preference shown for the epiglottis, more particularly when we are dealing with the very difficult task of distinguishing between syphilitic and tuberculous ulcers.

There is nothing pathognomonic in the appearance of syphilitic ulcers. It is true that Türck (Klinik, p. 301) claims sometimes to have observed ulcers which, with any considerable experience, one would recognize, at the first glance, as syphilitic. The characteristics of this "typical" ulcer, according to Türck, are "a more or less circular form, a deep floor, covered with a whitish-yellow coating, sharp, sometimes strongly elevated margins, surrounded by an inflammatory areola."

I confess that I cannot suppress strong doubts as to the "typical" character of this form of ulcer, and I must, for the present, attach myself to those authors—and they doubtless constitute a large majority—who do not recognize any characteristic appearance of the syphilitic laryngeal ulcer. At the same time, a sufficient number of fixed points, both numerically and in the matter of significance, still remain for diagnosis. Aside from the history of the case, we have the evidence of other syphilitic affections, especially in the pharynx, on the skin, in the bones; the preference shown by the ulcerative processes for the pharyngo-epiglottic ligament and the epiglottis; the presence of nodular infiltrations on the mucous membrane of the vestibulum laryngis; the swelling of the lymphatic glands at the angle of the jaw and in the submaxillary region; finally, the results of antisyphilitic treatment.

So far as the distinction between syphilitic and tuberculous ulcers is concerned, this can generally be established without difficulty, on the basis of the circumstances above mentioned and those spoken of under the head of Laryngeal Phthisis. The only serious difficulty presented, as a rule, is when a person who is suffering from old syphilis, especially of the pharynx, also has an induration at the apex of the lung, and suffers for some time with fever and hoarseness. In such cases all the ordinarily available data for a differential diagnosis leave us in the lurch, and the question has to be decided by the result of antisyphilitic treatment.

Terminations, Prognosis, and Treatment.

The extension of the ulcerative processes, where proper aid is not invoked, may involve the entire larynx and the trachea.

The destruction is often enormous, and those parts which are not deformed by the ulceration and the perichondritis finally succumb to the powerful traction of the cicatricial masses. Thus, as I have repeatedly observed, the epiglottis may be mowed down to a misshapen stump, the vocal cords may grow together, necrotic portions of the arytenoid and cricoid cartilages may be expelled, and the vestibulum laryngis may be so obstructed and grown together that the individual parts can no longer be distinguished.[1]

The result of such extensive changes, as a matter of course, is the hopeless ruin of the voice, and an interference with laryngeal breathing which imperatively demands the performance of tracheotomy, without giving any prospect that the tracheal canula can ever again be dispensed with.

This is the prospect which the *undisturbed* course of laryngeal syphilis opens, while, on the other hand, timely and energetic treatment can point to the most brilliant results. Even extensive defects, if they have not penetrated too deep, admit of complete cure, and even laryngeal perichondritis—otherwise the most dreaded result of laryngeal affections—need not cause the same alarm as under other circumstances. Those ulcers on the anterior surface of the arytenoids, accompanied with immobility of the latter and swelling of their investments, which are so suspicious in tuberculosis, are here of far less consequence, as has been truly remarked by Türck. As a rule, they may be entirely healed. Sometimes, even here, a lessened degree of mobility remains for a long time, if not permanently, but incurable necrosis of the cartilage is not so apt to follow as in tuberculosis.

The *syphilitic perichondritis* of the cricoid and the thyroid cartilages described by Dittrich (l. c.), Porter (l. c., cases 28 and 29), Ruehle (l. c., p. 277), Türck (Klinik, pp. 252 and 266), does, it is true, represent a much more serious affection, even in syphilis, than perichondritis of the arytenoids, and often leads to death, as the observations of the authors just named show. The fatal termination follows either through acute œdema of the larynx or through the results of the extensive suppuration of the perichondrium and the surrounding soft parts.

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[1] Türck has furnished us with numerous laryngoscopic pictures, coming under this category, in his Klinik.

The *fatal hemorrhage* from an extensive and deep ulcer of the region of the left vocal cord, observed in one case by Türck (Klinik, p. 413), may be mentioned as a rare termination of syphilitic laryngeal disease.

Finally, in speaking of terminations, mention may be made of the *papillomata*, which are fond of developing themselves in the vicinity of syphilitic cicatrices, and which, when they attain a great extent, may become hindrances to respiration and phonation.

In the matter of *prognosis* it is naturally a question of cardinal importance how far syphilis has progressed, and especially how far the ulcerative and perichondritic destruction has advanced at the beginning of treatment, and whether the constitution of the individual is adapted to energetic mercurial treatment. If the organism of the patient has been depraved by syphilis, or by repeated courses of mercurial treatment, to that extent that a fresh course of mercury cannot be carried out or is without results, the prognosis is rendered materially worse. Although some inveterate cases of syphilis are cured by the systematic use of the iodine treatment, yet this method does not possess anything like the degree of certainty that belongs to the energetic employment of mercury.

In *treatment*, the object by which we must be governed, at the outset, is to arrest, as speedily as possible, the progress of syphilis in the larynx. Every day may bring with it changes which not only derange the delicate mechanism of the larynx beyond cure, but may also directly threaten life (through perichondritis, œdema, or hemorrhage). Accordingly, not only the most speedy mercurialization of the system is necessary, but also local treatment which promises results. I agree with Sommerbrodt, Schnitzler, and others, in greatly preferring mercurial inunction to the internal administration of the remedy, and I institute inunctions twice a day, each time of a drachm of mercurial ointment. In the use of these strong inunctions the greatest care is to be exercised, from the beginning, to prevent stomatitis, which can almost always be done by washing out the mouth every half hour, using alternately fresh water and a solution of chlorate of potassa (twenty-five grains to the ounce), and frequently brushing the teeth with the same solution, accompanying this with mild laxative and diaphoretic treatment. Iodide of potassium (in a solution of from ten to fifteen grains to the ounce, a tablespoonful every two hours) I can only recommend in those cases where there is a depraved constitution, or where mercurial treatment does not produce the desired result, or, finally, when we desire—in the absence of any alarming conditions—first to establish the diagnosis of syphilis by the treatment.

For the *local* treatment of the ulceration, pencillings with diluted tincture of iodine or iodide of potassium in glycerine (Schnitzler), as well as with solutions of corrosive sublimate or nitrate of silver, are to be recommended. Painting the skin of the neck with tincture of iodine is also not to be omitted.

The effect of this general and local treatment shows itself very evidently even during the first days. No further extension of the disease takes place, the ulcers clean off, the secretion diminishes, the soft parts of the vestibule lose their swelling.

Of course therapeutics are powerless against necrosis and anchylosis of the cartilages, defects of the epiglottis, etc. Cicatricial deformities may be, at least partly, overcome with care: as, for example, the growing together of the vocal cords (Störck, Schnitzler), membraniform cicatricial bands at the laryngeal entrance, and extending from there to the pharynx, and others. The communications of Navratil, Schroetter, Schnitzler, Schech, and others, show how much can here be accomplished by skill and patience.

If the stenosis is such that not only the voice but breathing also is seriously interfered with, the attempt should still be made, after previous tracheotomy, which is generally rendered necessary by œdema of the larynx, gradually to free the passage again by the practice of dilatation. These attempts seem to be as difficult as they are fruitful, and under the untiring exertions of the laryngeal surgeons—I may mention von Bruns, Trendelenburg, Oertel, Störck, Schnitzler, and, above all, Schroetter[1]—begin of late to yield the finest results.

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[1] Compare Schroetter's newest work, "Beitrag zur Behandlung der Larynx-Stenose. Wien, 1876.

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Historical Sketch.

The history of neoplasms within the larynx, and of their successful treatment, does not begin until the sixth decennium of our century. Medical literature previous to that time contains only isolated observations of laryngeal tumors, which were either extirpated during life through the mouth or by opening the larynx, or were found on making autopsies.

The first extirpation of a laryngeal tumor is considered as having been performed by Koderik, in 1750, who operated, by means of what was called a rosary [Rosenkranz], on a laryngeal polypus, situated high up and capable of being reached through the mouth. The first description of laryngeal polypi, which caused death by suffocation and were not discovered until the post-mortem examination, appears to be that of Lieutaud.[1] Later observations of the same kind were made by Astley Cooper,[2]

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[1] "In cadavere cujusdam *asthmatici* triginta annorum, qui perpetuo querebatur de quodam impedimento in trachea, quod tussi et screatu expellere saepius conabatur et morte subitanea sublati, reperitur quidam polypus, variis radicibus laryngi infixus et versus glottidem obturamenti instar adactus, unde suffocatio inexpectata." (Observat. 63.) . . . "Secto cadavere cujusdam pueri duodecim annorum jampridem *phthisici* et inexpectata morte rapti, in propatulum veniebat intra laryngem corpus quoddam polyposum et racemosum tracheae superiori parte, pediculo unico et peculiari ortum trahens et hinc fluctans; quo forte ad laryngem repulso suffocationem obierat aeger." (Observat. 64.) *Lieutaud*, Historia anatomico-med. Lib. IV. 1767. Quoted in *Ehrmann*, Histoire des polypes du larynx. Strassburg, 1850.

[2] Quoted in *M. Mackenzie* (Growths, p. 5).

Otto,[1] Brauers (in Albers, l. c.), Regnoli,[2] Ehrmann, Rokitansky,[3] Horace Green,[4] Gurdon Buck,[5] Middeldorpff, Pratt (l. c.), Albers (l. c.), and others. Middeldorpff, in his work on the Galvanocautery, made a collection of sixty-four cases, and Lewin added sixteen cases more to the number. According to this, the amount of material, in the way of laryngeal polypi authenticated by literature in pre-laryngoscopic times, consists of eighty cases. But a very small number of these were correctly recognized during life, and of this number, again, but a small fraction was successfully operated on.

The situation changed materially on the introduction of the laryngoscopic mirror into the diagnostic-therapeutic armamentarium. Not only has the number of observations, within a short period (about seventeen years), exceeded four- or five-fold the sum of all cases belonging to pre-laryngoscopic times, so that the anatomical character, the seat and symptoms of laryngeal tumors now belong to the best known departments of laryngeal pathology, but the number of cures, through operative interference, by the path illuminated by the laryngeal mirror, has become larger than one would suppose. Since the first endolaryngeal extirpation of a polypus—accomplished by the perseverance of a von Bruns—the reports of favorable results following endo-laryngeal operative procedures have accumulated more and more, so that a collection of all the material in these reports is hardly any longer possible or of profit.

The uniformity in the clinical picture of certain neoplasms —for instance, the little fibromata of the vocal cords, and the almost invariably favorable result, under proper treatment, of the little operation for their removal—is no doubt the reason why the publication of such cases has of late become more and more rare.

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[1] Seltene Beobachtungen zur Anat. u. Physiol. Berlin, 1824. A man, sixty-five years of age, after being hoarse for several years, coughed up several "red flesh balls," the largest as large as a cherry, and finally died of suffocation. On post-mortem examination a number of tumors, of the size of a hazelnut, were found attached to the vocal cords by pedicles and obstructing the glottis.

[2] Osservazioni chirurg. etc. Pisa, 1836. Cited in Mackenzie. Regnoli removed a large neoplasm from the larynx, through the mouth, after previous tracheotomy.

[3] Zeitschrift der k. k. Gesellschaft der Aerzte in Wien. March, 1851.

[4] Polypi of the Larynx and Oedema of the Glottis. New York, 1852. Trans. of the Amer Med. Association. 1853.

[5] On the surgical treatment of morbid growths within the larynx, illustrated by an original case. New York, 1853.

It is an entirely different matter with those forms of neoplasm which, on account of their rarity as well as their significance for the life of the patient, demand the careful study of their details. To this class belong, among benignant growths, the papillomata, whose gravity consists in their extent and their eminent disposition to relapse; on the other side, carcinomata and the more rare tumors, such as the sarcoma, adenoma, chondroma, etc. Here we cannot but desire, for our information, the further collection of carefully reported cases.

Etiology and Pathogenesis.

Among the causes of the development of neoplasms in the larynx, *catarrh* heads the list, so far as benignant growths are concerned, and above all *chronic catarrh.* Accordingly all the injurious influences which may result in laryngeal catarrh are of decided influence in the production of laryngeal polypi. As these injurious influences especially reach the male sex, in consequence of its occupations, bad habits, etc., so laryngeal polypi, like chronic laryngeal catarrh, are found much more frequently among men than among women.

M. Mackenzie found among 287 cases of laryngeal neoplasm (exclusive of cancer), from his own practice and that of others, 197 in males and 90 in females; furthermore, 11.25 per cent. were among persons whose occupation led to the introduction of dust, smoke, and other irritants into the larynx. The mechanical irritations which the larynx experiences in occupations demanding loud speaking, crying, the giving of military commands (Mackenzie's "Out-door Occupations"), are decidedly favorable to the development of neoplasms.

At the same time, the greater liability of the masculine larynx may perhaps rest on other grounds than those of occupation alone, as, according to Causit, among forty-two little children who suffered with laryngeal neoplasms, twenty-eight were males and fourteen females.

In the matter of age, those of the middle class (from twenty to fifty years) are by far the most liable.

The tabular collection of Mackenzie's on this subject gives the following figures: From the 1st to the 10th year, six; from the 10th to the 20th, six; 20th to 30th, twenty-one; 30th to 40th, twenty-two; 40th to 50th, twenty-eight; 50th to 60th, fourteen; 60th to 70th years, three. Among 163 cases of other authors, in which the age was noted, 112 (that is, 68.7 per cent.) were from the ages of 20 to 50.

The period of early childhood was formerly regarded as possessing considerable immunity. On the other hand, Causit claims the specially frequent occurrence of laryngeal polypi during the first years of life, and even their occurrence congenitally. According to M. Mackenzie's report, among thirty-four preparations of the larynx, falling under this head, which he found in the various museums of London, not less than fifteen were of children under twelve years of age. Among twenty-four cases of papilloma, Oertel counted five under thirteen years of age.

Advanced age is decidedly favorable to the development of cancer in the larynx; still, in most cases, we lack any plausible explanation of why the cancer should localize itself just in the larynx. The statement of some authors that here, too, mechanical and other irritations of the laryngeal mucous membrane were of influence, cannot be considered as applicable, since the vocations which bring with them continued irritation of the larynx are by no means found to predominate among the subjects of laryngeal cancer. During younger years cancer is much more rare.

Pathology.

Anatomical Changes. Symptoms and Diagnosis.

A.—CONNECTIVE-TISSUE TUMORS.

1. The **Fibroma papillare,** or **Papilloma,** appears not only to occur the most frequently among tumors of the connective tissue, but to be the most common amongst all the new-formations within the larynx, if one may judge by clinical experience. Mackenzie counts sixty-seven papillomata among his one hundred neoplasms, twenty-nine of which were confirmed as being such,

after extirpation, by microscopic examination. Aside from the exciting causes—chronic catarrh, etc.—anomalies of constitution and of blood composition (chlorosis, anæmia, scrofula, tuberculosis) during the years of bodily development appear, according to Oertel, to have a favorable influence on the development of papillomata. The age of childhood seems to possess a certain predisposition to papillomatous development (Causit).

The vocal cords, and especially the anterior extremities of the same, are the most frequent seat and starting-point of papillomata. They are more rarely found on the epiglottis, the false cords, etc. Their capacity for recurrence is very great. When the extirpation of the tumors and the destruction of the subjacent tissue in which they originate is incomplete, they are generally developed again with considerable rapidity.

The development of papillary growths occurs, not only on the mucous surfaces originally provided with papillæ, but, as is especially shown by Virchow (Krankhafte Geschwülste. I. pp. 334 and following), also in regions where papillary structure of the mucous membrane is entirely lacking. The first step is the proliferation of the superficial connective tissue, the development of a little, amorphous, granular or homogeneous nodule, in which cells are not to be recognized till later. As the cells multiply, they gradually grow and put forth buds, just as is done by preëxisting papillæ (Virchow). The capillary loops of the papillæ are large; the surface of the neoplasm is covered with a thick layer of epithelial cells. The size and form of papillary tumors are very various. At first they represent little buttons or pegs; afterwards larger, warty formations, or those resembling a cock's comb; in case of luxuriant development,

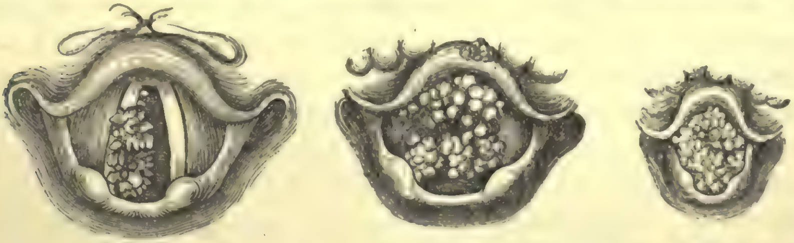
FIG. 17. FIG. 18. FIG. 19.

Various papillomata of the larynx. (After Oertel.)

huge growths similar to a berry, grape, or cauliflower in form, which may partly or entirely fill the upper and middle, more rarely the lower laryngeal cavity.

The laryngeal mirror usually gives quite satisfactory information with regard to the extent of the tumor, its point of origin, as well as the greater or less degree of vascularity of the neoplasm. The above drawings of Oertel may serve as types of the laryngoscopic images.

According to Oertel, the laryngoscopic image admits of our distinguishing three different forms of development.

The *first form*, and the least characteristic, consists of light-red or dark-red tumors, studded with more or less distinctly marked elevations, the main seats of which are the border, the under surface, or the anterior point of union of the vocal cords. They attain a size varying from that of a millet-seed to that of a bean, and are generally multiple.

The *second form* is that of a whitish-gray tumor of distinctly papillary structure. The arrangement of the rigid papillæ of which the growth consists is such that the larger groups are surrounded by smaller ones. They are almost invariably attached to the vocal cords by a broad base.

The *third form* consists of large reddish tumors, resembling a grape, mulberry, or cauliflower, which more or less completely fill the upper laryngeal cavity, and generally originate on the false vocal cords or in their vicinity (also on the epiglottis). The many-branching papillæ consist of a connective-tissue stroma, rich in cells and sap, and containing numerous thin-walled capillary loops.

The diagnosis of a papilloma can only be made with certainty by the laryngoscopic demonstration of the existence of this neoplasm in the larynx. The remaining symptoms, which are of clinical importance in so far as they give occasion to laryngoscopic examination, and eventually also to energetic interference (tracheotomy), are, above all, those of laryngeal stenosis. They consist of laryngeal dyspnœa, which is almost always mainly inspiratory; disturbances of the voice of various grades—according to Oertel, the voice seems to be most rough in the rigid papillomata of the margins of the vocal cords, especially if they extend far in the long axis of the cord; finally, a tormenting disposition to cough and the feeling of some foreign body sitting fast in the throat. The seat, form, size, and mobility of the papilloma in each individual case naturally determine the nature and intensity of the disturbance.

The broad and non-pedunculated papillomata that take root beneath the vocal cords may remain entirely without symptoms, aside from those caused by the primary chronic catarrh. Only when, as is often the case, they are situated immediately below the angle of union of the vocal cords, they may cause disturbances of voice by being partly squeezed between the cords when they are brought into juxtaposition.

A very instructive illustration of this was furnished me by a manufacturer, forty-nine years of age (Figs. 20 and 21). He had suffered from hoarseness for five years, and had tried, without any permanent benefit, the most various springs and bathing-places (Reichenhall, Ems, Weilbach, etc.), and had used all sorts of inhalations without effect. The mucous membrane everywhere was in a state of chronic catarrh.

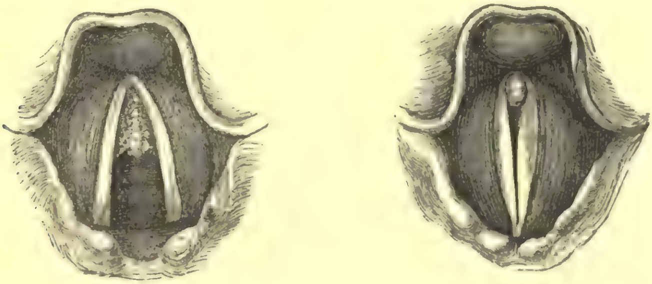

FIG. 20. FIG. 21.

Flat papilloma beneath the vocal cords. Fig. 20, On deep inspiration. Fig. 21, Partly embraced between the cords, in phonation.

Papillomata grow materially faster than most other tumors; in particular does the development of relapses after extirpation proceed with considerable rapidity—most rapidly, of course, in the large, grape-shaped, highly vascular papillomata, which, according to Oertel, may appear again within a few weeks after the operation. At the same time the primary papilloma seems generally to require some years for its development.

2. The **Fibroma, Fibrous Polypus of the Larynx,** is one of the more frequent forms of neoplasm in the larynx, though far less frequent than papilloma. Mackenzie found it in eleven per cent. of his cases; the youngest individual attacked was twenty-seven years old; the oldest, fifty-seven. The causes credited with its production are chiefly chronic laryngeal catarrhs and frequent mechanical injuries to the vocal cords, though individual predis-

position may also play some rôle therein. In contrast to the influence of anomalies of blood-composition in the pathogenesis of papillomata, Oertel calls attention to the perfect health of those individuals who are affected with fibromas.

The fibroma is very rarely multiple, and most frequently takes its point of origin on the vocal cords. Anatomically it presents itself as a little, generally pedunculated tumor, of a dirty whitish, or reddish, or dark-red color, sometimes with distinctly branching vessels on its surface. Its size seldom exceeds that of a hazelnut, though larger tumors have been observed in rare cases. As to outward form, the fibroma is usually roundish or pear-shaped, smooth or ridged. It may be hard or soft in consistence. Corresponding with this, its histological structure sometimes shows the characteristics of young and juicy, sometimes of older, tense connective tissue, the greater or less wealth in blood-vessels harmonizing with these conditions.

The growth of fibromata seems to be very slow; at least various authors, who have had the opportunity of observing such fibrous polypi during several years, have been able to recognize but a very slight increase in volume. So far as present observations go, fibromata have no tendency to relapse.

The vocal cords by far most frequently constitute the starting-point of fibromata—the upper as well as the lower surfaces of the cords furnishing the ground from which they spring. The mobility of the pedunculated tumors is of course greater the longer and thinner the pedicle is; the inspiratory and expiratory current of air slings these pedunculated tumors upwards and downwards, as is shown by the laryngeal mirror. The nodular

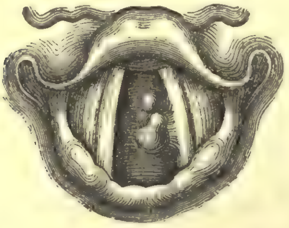

FIG. 22.

Knobbed connective-tissue tumor on the left vocal cord. (After Oertel.)

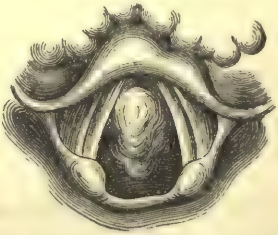

FIG. 23.

Connective-tissue tumor of the size of a hazelnut on the right vocal cord. (After Oertel.)

character of the tumors, which is tolerably frequent, may be recognized in the accompanying drawings of Oertel, of two different-sized fibromas.

I was enabled for several years to observe a fibroid, larger than a walnut, which was rooted to the upper (posterior) border of the cricoid cartilage, in the person of a mechanic in Erlangen, twenty-two years of age.

This tumor occupied the laryngeal entrance, in the manner shown in the accompanying picture, and yet, strangely enough, it impeded breathing so little that the patient persistently declined all operative interference. The disturbance to swallowing was, however, greater, and he was obliged to swallow very carefully in order to prevent the entrance of food (and especially of fluids) into the larynx. Furthermore, with every act of deglutition the larynx, in addition to its normal rising, made a movement forwards, inasmuch as at the beginning of swallowing the fibroid was crowded backwards and was hemned in between the plate of the cricoid and the vertebral column; the larynx, which was thus to a certain degree drawn up on the tumor, snapped back, downwards and backwards, at the end of each act of deglutition, with an audible noise.

As the patient afterwards died of acute nephritis, I had the opportunity of confirming the fibromatous nature of the tumor and its point of origin from the upper border of the plate of the cricoid cartilage (pharyngeal surface).

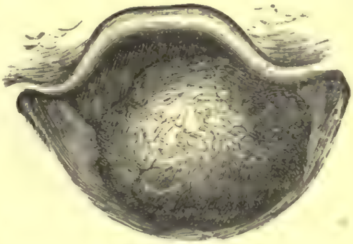

FIG. 24.

Fibroid at the laryngeal entrance, larger than a walnut, originating from the upper posterior border of the cricoid cartilage.

The functional disturbances in such cases are of course essentially different from those in tumors on or near the vocal cords. The latter, especially those that are pedunculated, if of large size, involve direct danger to life, inasmuch as they may be caught between the vocal cords and be the means of death by suffocation.

A patient of Lieutaud's—at the same time remarkable as being one of the oldest known cases of laryngeal polypus—who had for a long time felt a firm body in his air-tube and vainly labored to cough it out, died suddenly of suffocation on leaning out of bed to pick up a book that had fallen to the floor.

Smaller pedunculated tumors are also often caught between the edges of the vocal cords in phonation, and may thereby themselves receive a permanent impression if their position and attachment (short pedicle) is such that they are always pressed upon by the vocal cords in the same spot, or they may produce a permanent impression on the vocal cord of the opposite side.

Fibromas with a longer pedicle are only occasionally caught during the act of phonation; they are generally thrown out on to the upper surface of the vocal cords by the expiratory stream, and remain there till the next inspiration which draws them downwards again.

The accompanying laryngoscopic images (Figs. 25 and 26), which I obtained from a man thirty-six years of age, shows a polypus which was rooted by a broad pedicle below the left vocal cord, sinking down between the vocal cords on inspiration and resting upon them during phonation. The impression made on the corresponding point of the edge of the right vocal cord disappeared a few months after I had extirpated the polypus.

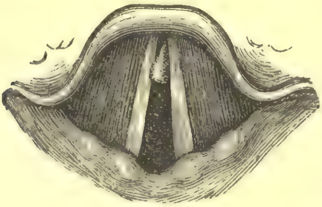

FIG. 25.

Pedunculated fibroma attached to the lower surface of the left vocal cord, as seen during inspiration.

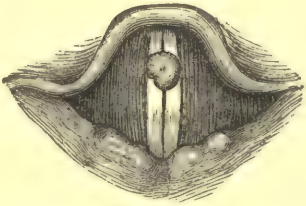

FIG. 26.

The same fibroma during phonation.

Quite similar pictures are given by von Bruns (Atlas, Plate VII. Figs. 10 and 18), Lewin, Türck, and others. Von Bruns' Case 1, which has attained historical reputation by being the first polypus extirpated through the laryngeal entrance (July, 1861), belongs in the same category (Atlas, Plate VII. Figs. 1 to 3). It was a large pear-shaped polypus which was attached by a broad pedicle below the left vocal cord.

The disturbances of the vocal cords vary very much, according to the seat, mobility and size of the fibroma, but are never entirely wanting when the fibroma is situated in the neighborhood of the glottis, if for no other reason, because the tumor maintains the mucous membrane for quite a distance around in a state of catarrhal irritation, which is liable to acute exacerbations through comparatively slight injuries (prolonged talking, taking cold). Little neoplasms may, nevertheless, be tolerated for years, even by those whose professions oblige them to talk (teachers, clergymen), without occasioning any special inconvenience—only that the voice, even in favorable cases, is rough, hoarse, and rattling, by reason of the polypus touching the vocal cords during the movements of phonation, and readily breaks into a falsetto by the formation of nodal points.

Relapses do not seem to occur after the extirpation of fibromata.

3. **Mucous Polypi** and **Cystic Tumors** are not exactly frequent in the larynx. They take root in the ventricle of Morgagni, by preference, being attached by a broad base. Growing gradually, they may protrude, as cystic polypi, far into the calibre of the upper laryngeal cavity. Virchow (Krankhafte Geschwülste, I. p. 246) gives a picture of such a pedunculated cystic polypus. The follicular cyst, which von Bruns (Laryngoskopie, XII. Beobachtung, Atlas, Pl. VII., Figs. 34 and 35) describes and pictures, comes under the same category. (This also took its origin in the ventricle of Morgagni.) The same is true of Gerhardt's colloid tumor of the vocal cord, Schroetter's and Mackenzie's cystic tumors of the epiglottis, and others.

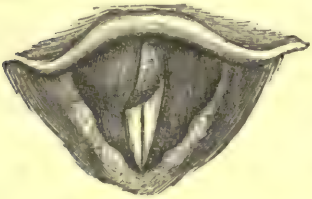

FIG. 27.

Mucous cyst of the ventricle of Morgagni. (After von Bruns.)

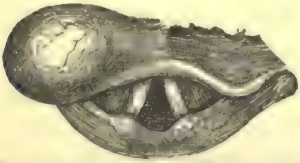

FIG. 28.

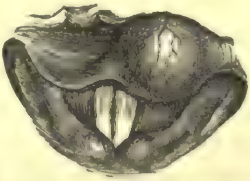

FIG. 29.

Cystic tumors of the epiglottis. (After Mackenzie.)

Mucous polypi and follicular cysts appear to grow slowly and seldom attain any considerable size, so that the functional disturbances caused by them are generally insignificant and confined to hoarseness. When incised, they slowly empty their more or less thickened contents. Their tendency to relapse, contrary to that of nasal mucous polypi, appears to be very slight. A free incision and subsequent thorough cauterization of the interior of what is left of the tumor, seems pretty certain to prevent a relapse.

4. **Myxomata** appear to be extremely rare within the larynx. One tumor, extirpated by von Bruns (Kehlkopfpolypen, p. 17), was pronounced by Schüppel to be a Myxoma hyalinum. Mackenzie observed a similar case (Growths, p. 48).

5. **Lipomata** seem to be the most rare of laryngeal growths. Only one authentic observation of this kind exists: that of von Bruns (Kehlkopfpolypen, p. 84), who extirpated a large (probably congenital) lipoma in a patient twenty-five years of age, by the galvano-cautery. The tumor was microscopically examined by Schüppel, and pronounced a lipoma.

B.—CARCINOMA.

Cancer occurs tolerably often within the larynx, in comparison to other neoplasms, being most frequently primary and confined to the larynx. More rarely it appears secondarily, extending to the larynx from the tongue, pharynx, œsophagus or trachea. The number of cases hitherto made public of primary laryngeal cancer—for it is of this only that we will here speak—is already pretty large. I have found ninety-six cases in all; still this may not exhaust the list. Ten of these were observed by myself.

We are entirely in the dark as to the causes of the occurrence of cancer in the larynx. In only one case (that of Démarquay, in Blanc, Obs. 4) did a traumatic injury—fracture of the thyroid cartilage from an attempt at strangling—precede the neoplasm by several months.

As in the development of cancer generally, so here also, *age* produces a material influence. Although laryngeal cancer may occur at any age, still the more advanced periods of life are far more liable thereto than younger ones. Among seventy-six cases in which the age was noted, I found

From 1 to 9 years of age	3	cases.
“ 10 “ 19 “ “	3	“
“ 20 “ 29 “ “	2	“
“ 30 “ 39 “ “	12	“
“ 40 “ 49 “ “	12	“
“ 50 “ 59 “ “	19	“
“ 60 “ 69 “ “	20	“
“ 70 “ 79 “ “	5	“

As regards *sex*, a very considerable majority are of the male sex. Among seventy-eight patients in my list, in whom the sex is given, sixty-two were males and only sixteen females.

No special influence can be assigned to any calling in life. In the list of cases, persons of every possible calling are to be found, no one predominating.

Among the different forms of cancer, the *epithelial* form is by far the most frequent. In sixty-eight cases on my list, in which the form of cancer was distinctly stated, fifty-seven were epithelial, nine encephaloid and scirrhous, and two villous.

The *seat* of cancer is almost invariably the upper and middle portion of the laryngeal cavity. It may originate in any part of this region, though the vocal cords and the ventricle of Morgagni seem to be the favorite points whence the evil spreads to the neighboring parts, upwards, especially to the false vocal cords and the aryteno-epiglottidean folds. The special liability of the vocal cords cannot, as yet, be proved, statistically, as, of the entire number of cases of laryngeal cancer observed, a number in itself not large, only those can be utilized for this purpose which were observed from the beginning, and in which the starting-point of the cancer was established. The number of these cases is, at present, however, too small to be of any value. Far more numerous are the instances in which definite information is given as to whether the cancer was of the right or left side or of both sides, multiple, or situated on the epiglottis alone. Among eighty-two such cases, we find the cancer situated

On the right side	17	times.
On the left side	19	“
On both sides, or multiple	33	“
On the epiglottis alone	13	“

The neoplasm either stands out, from the beginning, above the level of the surrounding tissue, as a sharply defined tumor, or appears more as a diffuse infiltration of the submucous layer, as a uniform or nodular intumescence of the soft parts, early interfering with the functions of the muscles, the cartilages, and the vocal cords, and narrowing the glottis.

As to their histological structure, the above-named forms of cancer show no deviation from the characteristics belonging to them when found in other localities.

The extent of the neoplasm, and the destruction caused thereby, may become very considerable if life is sufficiently prolonged, especially if the danger of stenosis of the glottis is removed by an early opening made into the upper air-passages. Not only the soft parts, but the cartilages too, are finally destroyed, either by the neoplasm itself or by the perichondritis excited thereby, which itself may again lead to extensive abscess-formation, necrosis of the cartilages, and a breaking through, outwards or inwards.

In the case of Désormeaux, in which, after the early performance of laryngotomy, life was maintained for over three years by the repeated extirpation of the neoplasm, as it relapsed, through the wound, post-mortem examination showed almost complete destruction of the soft parts and the cartilaginous framework of the larynx, especially its posterior portion, so that the cavity of the larynx opened, by a wide mouth, into the œsophagus; the upper tracheal rings also were destroyed, and only the epiglottis remained intact. At the same time there was no trace of infiltration of the lymphatic glands, neither was there any actual cancerous cachexia.

In a case observed by me, in which life was maintained for nine months after tracheotomy, the neoplasm crowded far into the trachea, so that finally, as in the case of Désormeaux, the introduction of the tracheal canula, when it was removed to be cleared, was rendered quite difficult by the presence of the neoplasm.

As a general rule, the ichorization of a cancer does not occur till pretty late. The same is true of the infiltration of the nearest lymphatic glands situated on the inner border of the sterno-mastoid muscle and beneath it. Generally, one single or several isolated hard glands appear at the side of the larynx after the cancer has existed for ten or twelve months, often even later; indeed, in some cases no infiltration of the lymphatic glands takes place.

I have found five such cases (among the above ninety-six) in which special mention was made of the absence of any swelling of the lymphatic glands. But perhaps they occur more frequently than might appear from this, as the presence of swollen lymphatics is only noted in seventeen cases.

At all events, the *early* involvement of the lymphatic glands—say within the first six months—is among the exceptions, a fact which is of great importance in the diagnosis of laryngeal cancer.

If the cancer is partly or entirely extirpated—which has been done repeatedly, both through the mouth and by laying open the larynx—the neoplasm pretty certainly recurs after weeks or months. Metastasis to other organs, however, does not seem to occur, or to be very exceptional, even on repeated extirpation and relapse of the growth. To judge by the few observations thus far on hand, total extirpation of the larynx does not appear to prevent a relapse in the neighborhood.

Symptoms, Diagnosis, Course, and Duration.

By far the most constant, the earliest and the most lasting of the symptoms of laryngeal cancer, is *hoarseness.* It generally precedes the other symptoms for a long time. sometimes for years.

In four cases in my collection the hoarseness existed for three years before the supervention of severer symptoms; in one case, four years; in two cases, five years; and in one case the patient had been hoarse for twenty-six years. A prodromal hoarseness of from one to two years seems to be the rule.

Other early symptoms are *pain in the larynx*, or in the depth of the *pharynx.* This often shoots out into the depths of one or the other *ear*, a manifestation which, according to my observation, may, under some circumstances, be of weight in making a differential diagnosis.

The complaint of darting or piercing pains in the ear was made to me six times in my ten cases, in part spontaneously and in part upon questioning, and indeed a few times in the absence of all other pain in the larynx or pharynx. In literature, I find ear-ache reported only three times; it is possible that it would have been found oftener if inquiry had been pointed directly towards it. In view of the difficulty of the diagnosis of laryngeal cancer, at the beginning, the presence of ear-ache might serve as a positive argument in favor of cancer, at least in view of the experience that it is but rarely met with in other affections of the larynx, excepting, of course, ulcerations. More numerous investigations directed to this point will have to establish the question of whether the significance really belongs to this symptom, which I am at present disposed to attach thereto. For that matter, I have found ear-ache a few times also in carcinoma of the œsophagus, seated high up (behind or under the cricoid cartilage), with constriction of the inferior laryngeal nerve.

I believe we may attribute the pain shooting out to the ear of the affected side to an irradiation of the irritation caused by the neoplasm in the sensitive fibres of the superior laryngeal nerve upon the auricular branch of the pneumogastric.

Sometimes diminution of strength and emaciation show themselves pretty early.

In chronological order, the *manifestations of laryngeal stenosis* are usually the next to appear. Laryngeal dyspnœa, at first insignificant and only appearing on considerable exertion, afterwards caused by every movement, finally becomes permanent, and generally shows itself more distinctly when the patient is lying on his back than in the upright posture—sometimes growing worse (especially at night), and again diminishing.

It soon becomes inevitable, in order to save the patient from suffocation, to make an opening into the upper air-passages. In the majority of the cases observed to the end, laryngo-tracheotomy was performed. Among the ninety-six cases of my collection there are thirty-eight in which the opening of the upper air-passages was undertaken, by one method or another. In a large number of cases the indefiniteness of the reports leaves us in doubt as to whether an operation may not, after all, have been performed at last.

If the disease is situated high up (epiglottis, aryteno-epiglottidean folds), *difficulty of swallowing* supervenes early, especially pain on swallowing and strangling. The same is true of *hemorrhages*, sometimes abundant, sometimes confined to bloody discoloration of the mucus expectorated. As the cancer breaks down on its surface, the hemorrhages are more frequent. The blood is mixed with pus or ichorous fluids, is fetid, and the breath, too, becomes foul. Single lymphatic glands at the inner border of the sterno-mastoid muscle swell, and the cancerous cachexia shows itself.

The manifestations of secondary *perichondritis*, if they appear early, may materially alter the picture of disease. The breaking through of abscesses, outwards, with subsequent profuse suppuration, may consume the vital forces more rapidly than the cancer alone would have done.

Laryngoscopic examination seldom shows very characteristic conditions at the beginning, especially in those cases in which diffuse cancerous infiltration into the submucous tissue produces a uniform intumescence of the soft parts, with strong, vascular injection of the mucous membrane. If the signs of perichondritis are early added to this, there may be great difficulty in establishing the differential diagnosis between primary perichondritis and primary cancer with secondary perichondritis. The recognition of cancer is easier when it appears in single nodules and ulcerates early, as is apt to be the case in the encephaloid form. But here, too, it is easy to confound the condition with syphilitic gummata, especially when it is well known that syphilis previously existed, even if extensive ulceration has already taken place.

The question is most simple in the circumscribed cancroid of the vocal cords. If a tolerably circumscribed neoplasm appears at this point, in a senile individual, after long-continued hoarseness or aphonia ; if, furthermore, there is pain in the ear of the corresponding side, laryngeal stenosis, emaciation, and loss of strength,—then, even without swelling of the lymphatic glands, which, as stated above, does not generally arise till late and is but slight, the diagnosis of carcinoma may be made, with tolerable certainty, from the start. This is particularly true if the neoplasm shows ichorization on its surface, and the symptoms of laryngeal stenosis of a higher grade than is ordinarily encountered in benign neoplasms of similar size.

If the process has advanced further, if the intumescence is very considerable, if there are extensive ulcerations within and enlarged lymphatic glands without, the diagnosis is no longer difficult. Neoplasms and destruction appearing at the same time in the pharynx or œsophagus, of course still further confirms it.

Laryngoscopic inspection gives such various pictures, according to the seat, extent, and histological composition of the neoplasm, that it is impossible to introduce *typical* images. I content myself with giving two laryngoscopic images, out of my own practice, together with reports of the cases, one of which was an epithelial carcinoma of the right vocal cord (about one year after its beginning), the other an epithelial carcinoma of the left vocal cord and ventricle of Morgagni, with perichondritis. (Compare Cases 2 and 3 of my article, mentioned above, in the Deutches Archiv f. klin. Medicin.

H. A., fifty-eight years old, foreman in a factory, admitted to the Medical Clinic at Erlangen, Dec. 2, 1873, has for the past three years found his voice to fail in loud calling and continuous speaking; for a year past has been hoarse, and has had occasional, and of late more lively, painful tickling in the right ear; still more recently dyspnœa on making an exertion—for instance, on climbing stairs. Emaciation and diminution of strength are very insignificant and have not been observed till lately. Twelve days before admission had a severe attack of dyspnœa lasting two hours, which has left a continued though moderate difficulty of breathing, which is aggravated by the air of a hot room and when lying on the back.

On admission the patient was moderately lean, not cachectic, almost voiceless, breathing with laryngeal stridor. Laryngoscopic examination showed a cauliflower growth on the right vocal cord, advancing pretty far into the cavity and crowding the right vocal cord, at its anterior extremity over towards the left. (Compare Fig. 30.)

The second night after admission the dyspnœa increased to the point of threatening suffocation, and demanded the immediate performance of tracheotomy. This was done by Prof. Heineke, followed by the best of results. Seven weeks afterwards the patient was dismissed to his home, still wearing the canula. Four weeks later he returned, requesting the performance of a radical operation. On the 26th of February, 1874, the larynx was laid open, and the neoplasm was scraped out with a sharp scoop. It was found to have entirely destroyed the right vocal cord. Microscopic examination showed it to be epithelial carcinoma. On the 18th of March, the wound was cicatrized. On the 25th the patient was discharged.

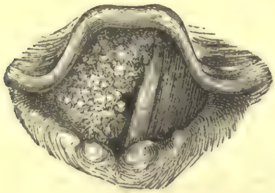

FIG. 30.
Epithelial carcinoma of the right vocal cord, about one year old.

Eight months after the operation (the 28th of October) the patient returned, with relapse. A large growth had pushed its way through, in front, and lay upon the thyroid cartilage, like an ichorous fungus. The glands of the neck were infiltrated. The entire upper portion of the laryngeal cavity now seemed occupied by the tumor, into which the entire posterior wall of the larynx was now merged.

On the 17th of November, the second removal of the tumor, by scraping it out, was undertaken, but could not be completely carried out on the posterior wall. The growth returned in the third week after the operation.

Death took place on the 23d of December, 1874, two years after the beginning of hoarseness.

Gottfried S., age fifty-five, a gentleman from Fürth, was received into the Medical Clinic at Erlangen on the 31st of May, 1872. He had previously always been well, being seized fifteen months before with hoarseness, and, as he stated, in consequence of taking cold, the attack coming on during one night, so that, while the evening before he could speak in a loud voice, he awakened the next morning entirely hoarse. This hoarseness, it is true, sometimes improved, but never disappeared altogether, and had increased of late to complete loss of voice. Aside from this, until within four weeks, the patient had made no complaint, especially not of any pain or swelling about the neck or in the throat, and considered himself perfectly well. Within four weeks, however, there had been difficulty of breathing, which had gradually increased, and produced suffocative attacks; furthermore, the patient had, within six weeks, observed a painless swelling on the left side of the larynx. There was no emaciation nor loss of strength.

Present condition: The man is very corpulent, and does not look cachectic. There is passive hyperæmia about the head and serious laryngeal dyspnœa. To the left of the larynx there is a tense infiltration of the soft parts, of the size of a walnut, penetrating into the depths. Puncture of the tumor, at its soft middle portion, yields no pus, only bloody serum. Laryngoscopic inspection reveals a flat, ulcerating neoplasm in the place of the left vocal cord, serious stenosis of the glottis and inflammatory œdema of the left false cord, the soft parts about the left arytenoid cartilage, and the left aryteno-epiglottidean fold. The conditions, otherwise, are entirely negative. No fever.

In the course of the next few days, the patient coughed up bloody mucus. The dyspnœa varied much in intensity, was greatest at night, accompanied by somnolency and cyanosis. On the 21st of June, after an absence of several days, the patient returned to the hospital, looking very badly and in great dyspnœa. As it was now no longer the flat carcinoma of the left cord, but the perichondritis of that side, or rather the œdema caused thereby, which seemed to produce the dyspnœa, the tumor found on the left side of the neck, alongside of the larynx, was incised, and on reaching the depth of an inch a large amount of chocolate colored, fetid ichor was evacuated. Great relief, and entire cessation of dyspnœa followed at once. But within a few hours later (eleven o'clock at night) severe dyspnœa returned, as well as an escape of fluid-blood by the canula, and clotted blood by the mouth. The immediate performance of tracheotomy (which he had hitherto refused) failed to prevent the fatal termination.

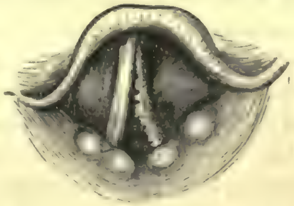

FIG. 31.

Epithelial carcinoma of the left vocal cord, with secondary laryngeal perichondritis.

Post-mortem (by Prof. Zenker) on the 22d of June, at 7 A.M. *Diagnosis on the Cadaver :* Carcinomatous ulcer of the larynx ; œdema of the glottis; caries of the ossified cricoid and thyroid cartilages. Large ichor-cavity, with hemorrhagic contents, on the left side of the neck. Parietal thrombus of the left internal jugular vein. Emphysema of the lungs. Small pneumonic infiltration of the lower lobe of the left lung. Slight sclerosis of the aorta. General adiposis.

That passage in the report of the post-mortem referring to the neck, is here given *in extenso.* Tracheotomy wound. Further, on the left side of the neck, a second incision, through which one reaches a wide, irregularly bounded cavity, reaching upward to the roof of the tongue and filled with blood-clots. Larynx and trachea crowded over towards the right. The epiglottis transformed, by enormous œdematous swelling, into a thick, translucent roll ; the œdema, furthermore, extends to the mucous membrane of the œsophagus and to the left aryteno-epiglottidean fold. The glottis is very greatly narrowed and slit-shaped. After laying open the larynx, the left half is seen to be occupied by an irregular tumor, beginning above in the pouch of Morgagni, and being from eight-tenths of an inch to a little over an inch in diameter. The edge of the ulcer is elevated, in part consisting of coarse papillæ, and in part simply thrown into hillocks. The base of the ulcer is very deep, covered with the remains of villous tissue. The cricoid and thyroid cartilages are to a great degree ossified, a large portion of the bony substance to the left being in part the seat of carious destruction, and in part infiltrated with a grayish-red, purulent mass. The tracheal mucous membrane is darkly injected, and of a slate color. The bronchi are occupied by larger blood clots.

The following case, which came under my observation, may serve to illustrate the difficulties which may attend the diagnosis, under some circumstances, and also go to prove that ear-ache may occur with any laryngeal ulceration.

H. A., age sixty-eight, a capitalist from Nürnberg, given to a cheerful enjoyment of life and noted for his loquacity, has for some months, without any demonstrable cause, experienced a pain in the upper half of the right thyroid cartilage, which radiates towards the right ear, and torments him day and night, being accompanied by a great disposition to swallow and an abundant and troublesome production of mucus. Of late there has been entire loss of appetite, emaciation, and loss of strength, hoarseness, and signs of laryngeal stenosis. He positively denies previous syphilis.

On examination, the patient appears lean and pale but not cachectic, has a small, soft pulse, moderate hoarseness, constant pain, a tormenting desire to swallow, in the region of the right upper border of the thyroid cartilage. There is moderate stridor on deep inspiration. No swelling of the lymphatic glands. Laryngoscopic examination shows a moderate-sized ulcerated surface on the right margin of the epiglottis, and in the right sinus pyriformis, serious, tumor-like intumescence of the right false cord, so that the right vocal cord is completely covered by it and the glottis appears somewhat narrowed. The mucous membrane of the larynx and the neighboring parts, on the right, is colored deep red and swollen. The surface of the ulcer is covered with an abundant muco-purulent secretion. No other anomalies to be found in the larynx, pharynx, or mouth, especially none leading to the suspicion of syphilis. No struma.

Although I almost always found myself compelled, by the laryngoscopic condition, the advanced age, the pain, the stridor, etc., to pronounce it a carcinomatous ulcer, yet, for the sake of safety, I ordered iodide of potassium internally and a solution of chlorate of potassa as a gargle.

After three weeks' use of the iodide of potassium the condition was materially improved, the ulcer almost healed. The injection and swelling of the soft parts had nearly disappeared, the pain and desire to swallow were reduced to a minimum, appetite had returned, and the appearance and freshness of the patient were about normal. After four weeks' further use of the iodide every anomaly had disappeared, except an insignificant cicatricial defect on the right border of the epiglottis.

Further observation, extending over nine months, showed that neither a relapse nor any other deviation from health occurred in the patient during that time.

After the brilliant results following the use of iodide of potassium, one cannot doubt in this case, that, in spite of all the declarations of the patient, we were dealing with a late, isolated outbreak of syphilis in the larynx and pharynx. Cases of this kind seem to justify the advice to try the use of iodide of potassium in every case in which the diagnosis is at all doubtful, especially with regard to distinguishing between carcinoma and syphilis.

The **course** of laryngeal carcinoma is, in general, very slow. In twenty-eight cases in which accurate information was given with regard to the duration of the entire disease, it lasted

From	3 to	6	months	4	patients.
"		9	"	2	"
"		1	year	3	"
"		18	months	7	"
"	over	2	years	2	"
"		3	"	3	"
"		4	"	3	"
"		6	"	1	"
"		10	"	1	"
"		15	"	1	"

Whether the histological character of the neoplasm has any influence upon its course, cannot at present be determined.

The early performance of laryngo-tracheotomy has a decided influence in prolonging life. In seven cases in which the duration of life, after artificial opening of the air-passages, is accurately noted, I find the minimum to be eight months and the maximum three and a half years, distributed as follows: in one case, eight months; in two, nine months; in two, one year; in one, fifteen months; in one, three and one-half years. It is true that the prolongation of his life is hardly a benefit to the patient, as the sufferings which the spreading and ichorization of the cancer bring with them are insupportable.

The *termination* is no doubt always fatal unless the entire larynx is extirpated. The assertion that extirpation of the neoplasm, with retention of the larynx, may lead to a permanent cure, as Schroetter claims to have observed in four cases, certainly requires further confirmation.

C.—OTHER TUMORS.

Sarcomata of the larynx are tolerably rare. In the literature of the subject, about a dozen cases are to be found[1] which have been confirmed by microscopic examination of portions of the tumors; still the number of observations may be larger. The nomenclature of the different forms of sarcoma is as yet so little established that no reliable statistics on this subject can at present be collected.

Sarcoma of the larynx, which almost always presents itself in the form of spindle-celled sarcoma (sometimes in the fibro-nuclear form), is generally seated on or near the vocal cords. The appearance of the tumor affords nothing characteristic; sometimes it is uneven, sometimes smooth, in color sometimes white, sometimes red. According to the observations now before us, the tendency to relapse is very great.

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[1] *Rauchfuss*, St. Petersb. med. Zeitschr. 1862. VI. p. 44. *Schroetter*, Wochenbl der Z. d. W. Aerzte, 1865. No. 34. *Oliver*, *Henry*, Amer. Journ. of Med. Sciences. 1867, p. 115. *Balassa*, Wien. med. Wochenschrift. 1868. No. 92. *Navratil*, Berl. klin. Wochenschrift. 1868. No. 49. *Gottstein*, Wiener med. Wochenschrift. 1868. No. 105. *Planchon*, Faits cliniques de la laryngotomie. Thèse. Paris. 1869. Obs. XVI. *M. Mackenzie*, Essay on Growths. 1871, p. 60. Tab. III. Figs. 3 and 7 (three cases). *Cohen*, Diseases of the Throat. 1872, p. 416.

Of **adenoma** only two cases have been published by Mackenzie, one case by von Bruns, and one by Cornil and Hérard.[1] They present no clinical peculiarities.

Angioma has only been observed in one case by Mackenzie (Growths, p. 53. Plate VI. Fig. 12). The vascular tumor was of the size and color of a blackberry, and was situated in the right pyriform sinus. With regard to the age and development of this tumor, nothing certain could be ascertained. Subjective annoyances had existed for six months.

Statements concerning **ecchondroses** of the laryngeal cartilages are made by Froriep, Virchow,[2] and Rokitansky. The statement of Ryland (l. c.) that he had seen "cartilaginous tumors" cannot be used, on account of the absence of any histological examination. The same is true of MacIlvain's[3] case of cartilaginous tumor frequently referred to by Urner, Albers,[4] and Froriep.

In a woman fifty years of age, who died of suffocation, MacIlvain found within the larynx a neoplasm of a hard, almost cartilaginous tissue, which nearly closed the chink of the glottis. When the tumor was removed, a little cavity was found, which was connected with a second tumor that lay outside of the larynx and attached to the thyroid cartilage.

This description gives no sure indication of the changes in the larynx. It might very easily have been an instance of the hard cicatricial remnants of laryngeal syphilis, as in the observation of Heine's, referred to below under the head of Resection of the Larynx.

We owe the first reliable observation of "chondroma laryngis" to Froriep.[5] Three, chiefly flat but pretty extensive, partly ossified tumors, arose from the inner surface of the thyroid cartilage, and had produced great narrowing of the laryngeal cavity.

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[1] *Mackenzie*, Growths, p. 52. *von Bruns*, Polypen des Kehlkopfes. 1868, p. 80. *Cornil* and *Hérard*, Sur la phthisie, p. 92.

[2] Geschwülste, I. p. 411. Also Deutsche Klinik. 1860. No. 46, p. 462.

[3] Edinb. Med. and Surg. Journ. Vol. XXXV. p. 215. 1831.

[4] *Gräfe* und *Walther's* Archiv f. Chirurgie. Vol. XXI. 1834, p. 532.

[5] Preuss. Vereinszeitung. 1834. No. 38. Also Schmidt's Jahrb. Bd. VII. 1835, p. 36.

Rokitansky,[1] too, observed partial outgrowths of the laryngeal cartilages, which presented themselves, on the *ossified* cartilages, as hyperostoses and exostoses.

Virchow describes the cartilaginous growths occurring on the cartilages of the larynx as sometimes diffuse and flat, sometimes more restricted and knotty. They are sometimes developed from the cricoid, sometimes from the thyroid cartilage, generally inwards towards the cavity of the larynx. Virchow found an ecchondrosis, covering the posterior half of the inner surface of the cricoid cartilage, two lines in height, and about the same in width at its base, and a larger one covering the anterior half of the inner surface of the thyroid cartilage. In the latter instance the growth was ossified, and constituted a true exostosis, under which the mother cartilage had maintained itself intact. Gintrac's description of a "concentric hypertrophy" of the cricoid cartilage (in Cruveilhier, Traité d'Anatomie pathologique, II. p. 274, 1852) is regarded with some distrust by Virchow.

In view of the rarity of ecchondroma in the respiratory apparatus, we may here, for the sake of completeness, refer to the beautiful case of broncho-stenosis ecchondrotica, published by Gerhardt.[2] The ecchondroma seemed to have arisen from frequent attacks of bronchitis and broncho-pneumonia, and had finally led to serious stenosis of the bronchial calibre.

With regard to the *diagnosis* and *therapeutics* of laryngeal ecchondroses, Virchow very rightly calls attention to the fact that, on laryngoscopic inspection, one might suppose that he had a polypus before him, as the growth is covered with mucous membrane; that, on the other hand, the firmness and hardness of these growths makes their operative removal from above utterly impossible.

Thyroid gland tissue as a neoplasm, producing stenosis in the lower laryngeal cavity, was recently observed by me in my Clinic. As this occurrence is thus far unique, so far as a careful search through literature permits one to judge, I will here briefly report the case.

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[1] Handb. d. pathol. Anatomie. Bd. III. p. 11.

[2] Jenaische Zeitschrift f. Med. u. Nat. Vol. III. p. 134.

J. G., a shoemaker, thirty years old, lay in my Clinic during the summer of 1875, with pleural exudation on the left side, which was absorbed after a long time with considerable shrinking of the lower lobe of the left lung and elevation of the diaphragm. After he had become perfectly able to work, he was seized, at the end of October, 1875, without any demonstrable cause, with laryngeal dyspnœa, which during the last week increased rapidly up to the point of threatened suffocation. On being admitted, there was moderate emaciation, severe laryngeal dyspnœa, loud stridor, great cyanosis, no pain in the larynx. Laryngoscopic examination showed the upper and middle laryngeal cavities to be intact, with normal structure and free mobility of the vocal cords. Nothing definite was to be recognized below the vocal cords. There was a moderately large bronchocele, which, it was thought, might press upon the trachea from behind. No other points for the explanation of the stenosis appeared.

During the next night there were suffocative attacks, still *tracheotomy* could be postponed till morning. The operation was rendered difficult by the condition of the thyroid gland, and the admission of air was not accomplished until the patient had ceased to breathe. Nevertheless, after performing artificial respiration for half an hour (mechanically and with electricity) life returned, and gradually intelligence too; still the pulse remained extremely frequent, and the respiration very irregular.

On the next day traumatic erysipelas set in with high fever, which carried him off on the day following.

The *post-mortem examination* (by Prof. von Buhl) showed the body rather emaciated; a tracheotomy wound above the manubrium sterni, the edges of the wound covered with yellowish-gray pus; the tissues of the mediastinum œdematous, and immediately beneath the manubrium infiltrated with pus.

The uvula, arches of the palate, and right tonsil were somewhat swollen, the thyroid gland not much enlarged, its tissue having in great part undergone colloid degeneration. Œsophagus and arch of the aorta normal.

In the larynx, beginning to the left of the middle of the cricoid cartilage, and extending below it for four-fifths of an inch in length, and two-fifths of an inch in thickness, there was a cylindrical tumor with a perfectly smooth surface, and covered with mucous membrane that was intact and somewhat injected. This neoplasm, as appeared from more careful, especially microscopic, examination, consisted of thyroid gland tissue. The bronchocele had pushed its way between the cricoid and thyroid cartilages on the left side into the lower laryngeal cavity.

The left lung, about its lower circumference, was firmly adherent, somewhat contracted and denser. The heart was covered with a thick layer of fat, its muscle rather softer than natural, a thrombus in the left ventricle extending into the origin of the aorta.

Here, therefore, death was caused by traumatic erysipelas and purulent mediastinitis, with the concurrence of moderate fatty degeneration of the heart. The thyroid-gland laryngeal tumor in itself would have justified a favorable prognosis.

Prognosis.

An absolutely unfavorable prognosis is afforded only by carcinoma of the larynx, and even for this a somewhat better prospect is offered by the recently accomplished complete extirpation of the larynx. The fact, that laryngeal cancer exists for an uncommonly long time as a purely local affection, and does not draw the lymphatic glands into sympathy until quite late, leaves room for the hope that radical cures will be possible in laryngeal cancer, as they have been observed in the early extirpation of cancers of the rectum, the penis, and the vaginal portion of the uterus.

It has already been stated above that early tracheotomy is capable of materially prolonging life in laryngeal cancer.

The prognosis of benign neoplasms is the better the more favorable the conditions for their operative removal through the pharynx. In large obstructing tumors, which demand previous tracheotomy, a certain degree of danger lies in this preventive operation. If, on the other hand, tracheotomy is refused or not undertaken, whether or not an attempt is made to reach the tumor through the mouth, the danger of suffocation grows in proportion to the voluminousness and vascularity of the neoplasm, or in proportion as the pedunculated character of the tumor increases the possibility of its becoming suddenly caught in the glottis and producing suffocation. The number of cases that have perished in this way is tolerably large, according to the statements made in literature; undoubtedly, however, this termination has become much less frequent since the introduction of laryngoscopic surgery.

Among the benign tumors, which have become the subjects of modern operative interference, papillomata justify a less favorable prognosis than connective-tissue tumors, owing to the great tendency to relapse in the former, and to their generally large size. The most favorable prognosis is afforded by the little pedunculated fibromata and mucous polypi, which are easy to extirpate and do not recur.

Therapeutics.

As in neoplasms of the larynx all disturbances depend entirely and immediately upon the neoplasm itself, the therapeutic indications are very simple, *to wit*, the most complete possible removal of the neoplasm and the prevention of relapses; or, if the operative removal of the growth is impossible or practicable only in part, the prevention of the dangers which the unlimited extension of the tumor within the larynx would bring with it; or, finally, if the neoplasm has been positively determined to be cancer, the early extirpation of the larynx.

The radical operation for the removal of laryngeal neoplasms may be undertaken by different ways and in different manners.[1]

The Endolaryngeal Methods of Operation.

The removal of the neoplasm through the mouth and pharynx, "per vias naturales," guided by the mirror, is the simplest and the safest, if the necessary precautions are observed. Since the famous first extirpation of a pedunculated fibroma, which von Bruns undertook in the case of his brother with the finest result, this method has been practised in hundreds of cases, and yields the best average results for the functions of the larynx, that is, for its *restitutio ad integrum.* It will always be preferred to the other more formidable methods of operation, wherever there is a possibility of carrying it out.

For the removal of neoplasms by this way various methods have been proposed and numerous instruments constructed.

Removal by Cauterization is adapted only for small, soft polypi and papillomata above or below the vocal cords, whether of primary or of secondary origin (for instance, in the vicinity of phthisical or syphilitic ulcers).

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[1] Regard for the amount of space allowed naturally forbids entering in detail on the individual methods of operation. We must confine ourselves to treating in general of the methods, their indications, and their results, and refer our readers for details to the special works of von Bruns, Mackenzie, Lewin, Tobold, Schnitzler, Schroetter, Navratil, Oertel, and others.

I have removed a certain number of little polypi and papillomata through cauterization with nitrate of silver in substance, and believe I ought to recommend the trial of cauterization, especially in persons afraid of the knife. If the little growth does not disappear under cauterization, then the latter has at least prepared the way for the introduction of cutting instruments.

The best caustic to use is *nitrate of silver*, and the best method of application is by the flexible sound, ending in a little roughened knob or plate, as first introduced by Lewin. The roughened end of the sound is lightly dipped into the fused nitrate of silver (held over the flame of a lamp in a porcelain vessel), and thus becomes covered with a thin but closely adherent coating of the caustic, quite sufficient for *one* cauterization. The complicated covered *porte-caustiques* (of Stoerck, Rauchfuss, Türck) seem on the whole to be superfluous.

Chromic acid is preferred to nitrate of silver by some (Tobold) in those cases where it is a question of more energetic and deeper cauterization of the remains of a tumor, or its point of origin, for instance, after the removal of papillomata. The introduction of the readily deliquescent crystals of chromic acid must of course be accomplished by means of a covered holder, and requires great caution.

The immediate result of cauterization with nitrate of silver or chromic acid is a severe spasm of the glottis with a sense of suffocation, which, however, is accompanied by no danger, but usually ceases in the course of a few minutes and after a drink of cold water, leaving behind it a burning pain, active secretion of mucus, and intense hoarseness. The latter manifestations, as well as the reactionary inflammatory swelling, disappear of themselves in a few days with the throwing off of the eschar. The cauterization of neighboring, sound portions of mucous membrane, which is often inevitable, as they are pressed forward by the spasmodic action, is of no consequence.

Removal by Cutting Instruments.—In all larger and more resisting neoplasms cutting instruments are indispensable. The choice of the instrument, whether knife, scissors, guillotine, or wire loop, depends on the requirements of the individual case.

The *lance-shaped knife* is the one best adapted to the cutting off of pedunculated polypi, especially those situated on the margin of the vocal cords, as well as to cutting into neoplasms that are attached by a broad base. In smaller, non-pedunculated polypi, as I have often convinced myself, repeatedly slicing them off and cutting into them is sufficient to insure their being thrown off. After repeated incisions, the polypus, through hemorrhagic infiltration, turns of a bluish-red, gradually becomes necrotic, and imperceptibly disappears.

The best plan is to supply one's self with a number of lance-shaped knives of different sizes, and with the double-edged end-piece in different positions, so as to be able to cut off the largest and the smallest pedicles as well as to be able to make incisions both longitudinally and transversely. It is also desirable to have some probe-pointed knives (von Bruns, Tobold), a part of which should have their cutting surface on the convex and a part on the concave side. Concealed knives may, in general, be dispensed with. They have this great disadvantage, that the delicate guidance of the instrument is interfered with by the drawing back of the guard or cover, or the pushing forward of the knife; besides which, any tolerably skilled laryngo-therapeutist will be able to avoid wounding the surrounding soft parts without a cover to his knife, especially if, in case of special irritability of the mucous membrane, the patient is made the subject of sufficient previous practice in the introduction of the sound.

Instruments in the form of *scissors* (von Bruns), of *fenestrated knives* (Türck), of *laryngeal guillotines* (Voltolini, von Bruns), which cut with a ring-shaped knife on the principle of Fahnenstock's tonsillotome, of *concealed knives* (Türck), etc., are probably not very often used. By their volume they too greatly limit the already restricted space, and have no real advantage over the simple lance-shaped knife or the wire snare.

The *wire snare* or *laryngeal écraseur* has found a more extensive application of late, and may be designated as the instrument which acts in the quickest and most thorough manner and with the least danger to neighboring parts, for the removal of larger neoplasms (fibromas, papillomas), when not attached by a very broad base. Among the holders of the wire loop constructed by Gibb, von Bruns, Tobold, and Oertel, the écraseur of Tobold (compare Fig. 32 *a*) and of Oertel deserve the preference, because the wire loop can be drawn so far back into the holder that the very last fibres of the pedicle are cut off. Throwing the loop over the polypus and the manipulations to make it take hold close to the laryngeal attachment of the pedicle, so as, if possible, to cut off the entire pedicle, of course require great skill, which can only be attained by practice. Fine, annealed iron wire is the best and cheapest material for the snare (Oertel).

Removal by Means of Forceps and Instruments for Crushing.—Polypus-crushers (Türck) or laryngeal forceps (von Bruns, Lindwurm, Stoerck, Tobold, Oertel) are adapted only to those cases in which soft polypi, attached by slender pedicles, can easily be seized and be caused to mortify by being crushed. As Tobold has justly declared, the actual twisting off or tearing out of the neoplasm is not advisable, on account of the possible lesions that might be produced in neighboring organs, and is only permissible when the pedicle is already thinned by having been previously cut into, or when the entire mass of the tumor is very soft. Figure 32 *c* represents Oertel's laryngeal forceps-holder, the closure of the forceps being very easily and conveniently accomplished by means of a lever. A large number of points have been constructed to fit into this holder, constituting in part cutting and in part crushing instruments, sufficient, probably, to satisfy all demands.

It may be remarked, by the way, that various sizes of laryngeal forceps should be found in the armament of every physician, because it is only by their means that one is enabled to effect the safe and speedy removal of foreign bodies (fish-bones, splinters of bone, needles, etc.) that may be caught in the laryngeal entrance.

Removal by Means of the Galvano-cautery and Electrolysis.—The galvano-caustic loop possesses only this advantage over the simple wire écraseur, that all hemorrhage is avoided, which certainly is a matter of some consequence in highly vascular neoplasms. Furthermore, the galvano-cautery may be made to penetrate quite energetically into the depths, if we desire to destroy the base of an extirpated papilloma or sarcoma, and thus endeavor to prevent local relapses. Aside from this, the galvano-cautery has no advantages, and is therefore, as it appears, but rarely employed of late.

Voltolini, von Bruns, and Schnitzler, in particular, are worthy of credit for having developed the different galvano-caustic methods of operation.

The destruction of neoplasms by *electrolysis*—that is, by the decomposition of the salts of the blood-serum through the galvanic current, and the caustic action of the alkalies at the negative pole—has also been recommended for the destruction of laryngeal tumors, according to the experiments of Althaus,[1] von Bruns,[2] and, of late, amongst others, of Fieber. Fieber sinks a needle, guarded by a hard rubber tube and connected with the negative pole of a battery, into the neoplasm, while the positive pole completes the circuit at some point on the surface of the body. The caustic action of the alkalies upon the tissue of the neoplasm produces a grayish-white eschar around the negative pole, as represented by the needle, without causing the least hemorrhage. After this process has been repeated several times, the tumor shrivels up, according to Fieber, whereupon the rest may be removed by cauterization with nitrate of silver. This procedure, which thus far does not appear to have been tried by other authors, does not seem to possess any special advantages over the thermal galvano-cautery and the methods of removal by cutting and by the écraseur—indeed, it seems to involve a good deal of trouble. Nevertheless, it is desirable that it should be thoroughly tested by others.

II. Extirpation through the Neck.

In opposition to the former teaching on this subject, Planchon has proved that **thyreotomy**, or the operation of laying open the larynx, was first undertaken by Pelletan in 1788, for the removal of a foreign body, and was repeated by Blandin in 1828, for the same purpose. For the purpose of removing a neoplasm from

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[1] A Treatise on Medical Electricity. III Edit. London, 1873.

[2] Die Galvanochirurgie oder die Galvanokaustik und Elektrolysis bei chirurgischen Krankheiten. Tübingen, 1870.

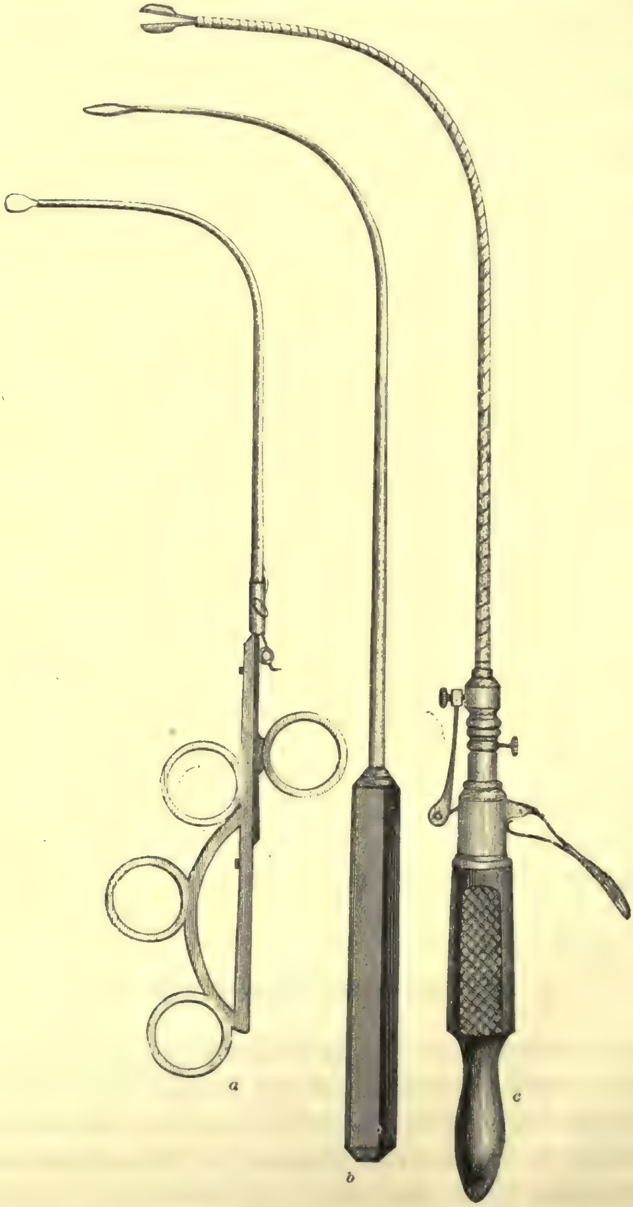

FIG. 32.

a, Écraseur. (After Tobold.) *b*, Lance-shaped knife, with double cutting edge. *c*, Laryngeal forceps. (After Oertel.)

the larynx, thyreotomy was first successfully carried out by Brauers, in Löwen, in 1833. Then follow two operations by Vital (1838) and Maisonneuve (1839) for foreign bodies; and finally the celebrated thyreotomy of Ehrmann (1844), in Strassburg, for the extirpation of a laryngeal polypus, which has hitherto had the credit of being the first.[1]

Since the introduction of laryngoscopy the number of thyreotomies has rapidly increased, as the laryngeal mirror has afforded the opportunity for more accurate diagnosis and for clearer indications for the operation. In the aggregate, I find forty-five cases of thyreotomy on record, including the American observations given by Beschorner in the appendix to his essay,[2] as well as the case of epithelial cancer narrated above, which was observed by me, and in which Prof. Heineke did the thyreotomy.

If we except the removal of foreign bodies, the indication for thyreotomy is confined to the extirpation of laryngeal neoplasms which cannot be removed, or at least not radically extirpated, through the mouth—as, for instance, cancers, sarcomata and chondromata, as well as to the removal, perhaps, of cicatricial masses producing stricture.

The operation, though not exactly dangerous to life (the number of deaths following the operation seems hardly to reach five per cent.), is yet a severe one and followed by serious consequences. The necessity of previously performing tracheotomy, in order to be able to operate under chloroform without danger of suffocation, and with the assistance of Trendelenburg's tracheal canula, as well as the usually persistent disturbance of voice, a result of the almost inevitable wounding of the vocal cords—that is, of their anterior insertion—or the distortion and displacement of the soft parts in consequence of cicatricial contraction, naturally, in every case, awaken serious scruples with regard to undertaking this operation. Still these scruples cannot be allowed to prevail over against the severe disturbances which urge the performance of thyreotomy, inasmuch as they are totally incurable by any other means.

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[1] Compare *Hueter*, Tracheotomie und Laryngotomie, l. c.

[2] D. Zeitschr. f. klin. Chirurgie. 1873, II. p. 471.

With regard to the details of the operation, I must refer the reader to treatises on surgery, especially to the above-mentioned work of Hueter.

The Opening of the Air-passages above the Larynx—more correctly, opening of the pharynx—*pharyngotomia subhyoidea* (von Langenbeck), *laryngotomie sous-hyoidienne* (Malgaigne)—is entitled to mention here, because this operation has repeatedly been undertaken for the removal of neoplasms which were attached within the laryngeal entrance or in the vestibule.[1]

Previous tracheotomy and the use of the tampon canula facilitate the operation for both operator and patient, permitting the use of chloroform, and, by preventing the entrance of blood into the trachea, rendering the operation, which of itself is hardly at all dangerous, quite free from danger. In view of the modern development of laryngoscopic and laryngo-surgical art, however, the necessity for the employment of this operation for high-seated laryngeal neoplasms is reduced almost to zero.

III. Extirpation and Resection of the Larynx.

The *total extirpation of the larynx* was first carried out by Billroth, in 1873, for carcinoma, after Czerny,[2] by a series of experiments on animals, had demonstrated the feasibility of the operation, and the possibility of having a canula retained within the wound in the place of the larynx.

Billroth's patient, in whom the larynx was successfully extirpated, not only had his life saved, but, by means of an artificial vocal apparatus constructed on the plan proposed by Gussenbauer, received the faculty of speaking again, in an audible though monotonous voice.

The second extirpation was undertaken by Heine, also on account of carcinoma, and likewise terminated successfully, at least so far as the operation is concerned. Death followed six months later from a relapse of the cancer.

A third case was also operated on for cancer by M. Schmidt, in Frankfurt. The patient died on the sixth day after the operation.

The fourth extirpation was undertaken by Bottoni, in Novara, on account of a neoplasm, and terminated successfully—that is, four weeks after the operation the patient was in as good condition as could be desired.

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[1] *Pratt*, Gazette des Hôpitaux, 1859, No. 103. Schmidt's Jahrbb. Bd. III. p. 226. *von Langenbeck*, Berliner klin. Wochenschrift, 1870, No. 2.

[2] Wiener med. Wochenschr. 1870, Nos. 27 and 28.

In the fifth case von Langenbeck removed not only the entire larynx, but also the hyoid bone, a part of the tongue, of the pharynx, and of the œsophagus, on account of carcinoma. On the seventh day the patient was free from fever. The final result is not yet known.

A sixth operation of this kind was recently performed by Billroth, but has not yet been published.

The foregoing six cases do not, it is true, determine the question of the value of total extirpation, but they certainly justify the conclusion that this radical operation has a future before it, in carcinoma and other severe alterations in the larynx, incapable of recession. The dangers which attend the operation are not so great as to hinder us from exposing a patient to them, who is in any event doomed to death. In future cases the endeavor should be to accomplish the extirpation at the earliest possible stage, as soon as the diagnosis is fully established, because thereby the prospect of avoiding relapses will of course be improved.

In syphilis, and other affections causing permanent deformity of the larynx, *resection of the larynx*, as first performed by Heine, might well be preferred to total extirpation, as this operation is less extensive than extirpation, affects the swallowing apparatus less, and renders possible the restoration of laryngeal breathing and of the voice (by inserting a tube, which at the same time contains a vocal apparatus, into the gutter remaining at the site of the larynx).

This "*subperichondrial resection*" of the lateral portions of the thyroid and cricoid cartilages was first carried out by Prof. Heine, in December, 1874, on account of extreme laryngeal stenosis, due to a sclerosed chondro-perichondritis—as it afterwards appeared, of syphilitic origin. The cartilaginous lateral portions of the laryngeal framework were removed, beneath the perichondrium, by means of the bone forceps. The result of the operation was at first very favorable; the patient could not only breathe through a canula laid in the remaining laryngeal gutter, but could also speak through a vocal apparatus placed within this canula.

The patient succumbed a year later to pulmonary phthisis, together with a severe relapse of syphilis in the pharynx and larynx.

The verdict with regard to this new operative procedure, and especially with regard to its practical value, must be left to the future.

THE NEUROSES OF THE LARYNX.

Anatomico-physiological Introduction.

It is well known that the foundation of our present knowledge of the origin and function of the laryngeal nerves was laid by Longet, although in some respects extended and more firmly established by later investigators, especially Bischoff, Rosenthal, Luschka, Schech, Heidenhain, Bernhard, and others.

The superior and inferior laryngeal nerves, branches of the pneumogastric, are to be regarded as the only innervators of the sensitive and motor tracts of the larynx. And yet only the sensitive fibres to be found in them are true offshoots of the pneumogastric ; the motor fibres of both laryngeal nerves are only mingled with those of the pneumogastric, and originate in the spinal accessory nerve of Willis.

The sphere of action of the two laryngeal nerves is limited as follows :

The **superior laryngeal nerve** supplies sensibility to the upper and middle laryngeal cavities, down to the border of the vocal cords, as well as to the contiguous portions of the pharynx and the root of the tongue. It further supplies motor filaments to the crico-thyroideus muscle, and in man probably also to the thyro-epiglottidean and aryteno-epiglottidean muscles.

The superior laryngeal nerve, as is well known, arises from the pneumogastric at the lower end of the plexus nodosus, passes down in front of the superior cervical ganglion of the sympathetic, then between the internal and external carotids obliquely forwards and downwards, and, reappearing from behind the external carotid, divides at an acute angle into its internal and external branches.

The external branch, accompanied by a sympathetic twig from the superior cervical ganglion, supplies the lower constrictors of the pharynx, the thyro-pharyngeus and crico-pharyngeus muscles, and ends in the crico-thyroideus muscle. It is entirely or chiefly motor.

The internal branch, considerably larger than the external, penetrating the hyothyroid membrane, enters the pharyngo-laryngeal sinus, and running diagonally through the same, immediately under the mucous membrane, divides into several twigs, which, according to Luschka, in part penetrate the muscles (for instance, the arytænoideus muscle), and finally is distributed to the mucous membrane of the upper laryngeal cavity and the epiglottis. It is still a mooted question whether or not the motor innervation of the arytænoideus and the depressors of the epiglottis (the thyro-epiglottidean and aryteno-epiglottidean muscles) is derived from the internal branch. Luschka at first accepted the affirmative side of the question,[1] but gave it up again in one of his more recent monographs on the anatomy of the larynx,[2] and attaches to the filaments entering the muscles only the significance of perforating sensitive branches distributed to the mucous membrane. The complicated nature of the anatomical relations, especially the circumstance that many sensitive fibres penetrate the muscles in order to reach the mucous membrane, does not permit us to expect that this question will be settled by the anatomist's knife; the innervation of the muscles in question will have to be settled by experiments on animals and by clinical observation. The more recent investigations, however, on the function and innervation of the laryngeal muscles—in particular, the careful and circumspect experiments of Schech—determine nothing on this point. Clinical observations, especially the observations of Kappeler after section of the pneumogastric, as well as the occurrence of diphtheritic paralysis of the depressors of the epiglottis, in connection with paralysis of the crico-thyroideus muscle and anæsthesia of the laryngeal mucous membrane, while all the rest of the muscular apparatus was intact (these observations will be communicated further on), seem to me to argue in favor of Luschka's first opinion, in which Gerhardt also agrees, that in man the innervation of the depressors of the epiglottis depends on the superior laryngeal nerve.

The innervation of the crico-thyroideus muscle by the external branch of the superior laryngeal nerve is universally recognized. The doubts recently cast upon this by Navratil cannot, according to the criticisms of Schech (l. c., p. 271), be looked upon as well grounded, because the method of operation which Navratil claims to have followed in his experiments leaves it open to very serious doubt whether he really cut the internal branch of the nerve.

Schech, by his method of operation, obtained quite uniform results, as follows: section of the external branch of the superior laryngeal nerve, or of this nerve itself, hindered the longitudinal tightening of the vocal cords by paralysis of the crico-thyroideus muscle, was followed by a rough and deep voice, and made the production of high tones impossible.

Türck believes, according to clinical observations, that we must hold to a double innervation of the crico-thyroid muscle, referring it to both the superior and inferior laryngeal nerves; or, at least, that we must suppose trophic fibres to be supplied by the recurrent laryngeal, because in simple paralysis of the recurrent (the superior laryngeal being therefore intact) he has repeatedly found the crico-thyroid muscle to be atrophied. At all events, it may be well hereafter to pay attention to the point thus brought up by Türck, especially in post-mortem examinations of the frequent cases of unilateral paralysis of conduction of the recurrent laryngeal nerve.

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[1] Die Anatomie des Menschen. I. 1 (Hals). p. 286. Tübingen, 1862.

[2] Der Kehlkopf des Menschen, p 166, Tübingen, 1871.

The same attention should also be directed to the condition of the arytænoideus muscle in paralysis of conduction of the recurrent laryngeal, as this muscle, too, has been sometimes credited with receiving its innervation from both laryngeal nerves, and sometimes with receiving it from the inferior nerve alone. The question is far more difficult to determine here than in the crico-thyroid muscle—at least, for clinical and pathologico-anatomical investigation—because the arytænoideus does not exist as a pair, and, in case of unilateral paralysis, its innervation might, perhaps, to a certain degree be vicariously assumed by the nerve of the other side.

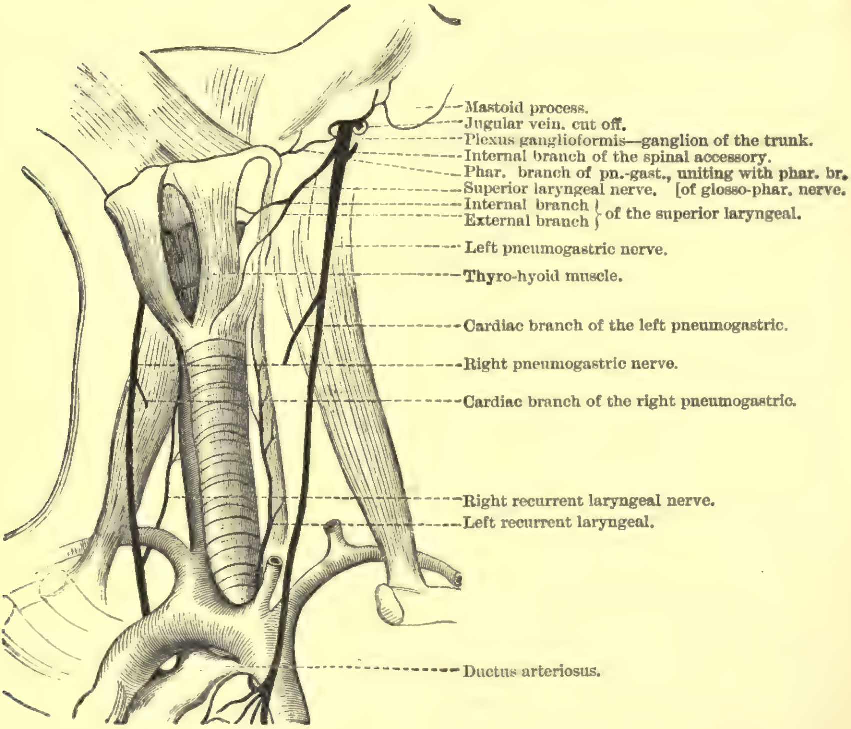
Mastoid process.
Jugular vein. cut off.
Plexus gangliiformis—ganglion of the trunk.
Internal branch of the spinal accessory.
Phar. branch of pn.-gast., uniting with phar. br.
Superior laryngeal nerve. [of glosso-phar. nerve.
Internal branch
External branch
of the superior laryngeal.
Left pneumogastric nerve.
Thyro-hyoid muscle.
Cardiac branch of the left pneumogastric.
Right pneumogastric nerve.
Cardiac branch of the right pneumogastric.
Right recurrent laryngeal nerve.
Left recurrent laryngeal.
Ductus arteriosus.

FIG. 33.
The laryngeal branches of the pneumogastric nerve in the newly born subject. (After Henle.)

With regard to the *function* of the muscles hitherto named, it may be briefly stated that the thyro-epiglottideus and arytæno-epiglottideus muscles draw the epiglottis downward, and that therefore, when these muscles are paralyzed, the epiglottis must stand upright and immovable, resting against the root of the tongue.

The function of the arytænoideus muscle is the drawing together, the "juxtaposition," of the arytenoid cartilages, whereby (especially with the help of the crico-arytænoidei laterales and the thyro-arytænoidei muscles) the vocal processes are also drawn nearer together. Paralysis of the arytænoideus muscle must therefore be followed by gaping of the cartilaginous glottis on attempts being made at phonation.

The function of the crico-thyroidei muscles is, in several respects, still a matter of controversy, yet it may at present be considered as most probable that these muscles either draw the anterior portion of the cricoid cartilage upwards against the thyroid cartilage, which has been fixed by the hyothyroid muscles, and at the same time push the entire cricoid cartilage somewhat backwards (musculi crico-thyreoidei obliqui of Henle); or, reversing this action, that the thyroid cartilage is drawn down against the cricoid, and at the same time pushed somewhat forwards. In the first case, which, according to the experiments of Jelenffy and Schech, is the most probable, the plate of the cricoid is tipped somewhat backwards through turning of the cartilage in the crico-thyroideal articulation, and thus the arytenoid cartilage, seated upon the upper margin of the plate, and fixed in its position by the posterior and lateral crico-arytenoid muscles, and the arytænoideus, is displaced further to the rear, and therewith removed from the anterior angle of the thyroid cartilage. At all

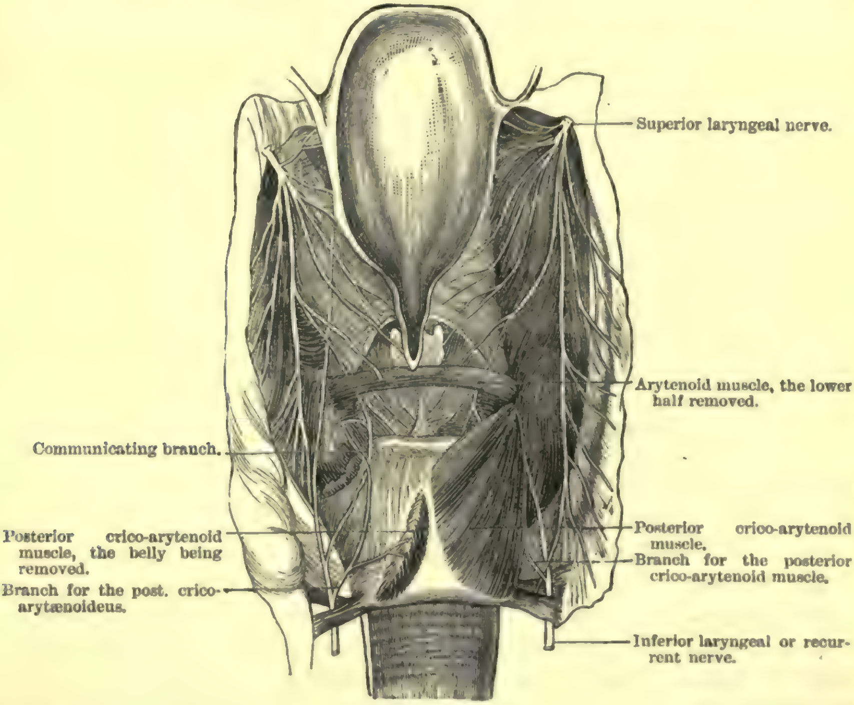
Superior laryngeal nerve.
Arytenoid muscle, the lower half removed.
Communicating branch.
Posterior crico-arytenoid muscle, the belly being removed.
Posterior crico-arytenoid muscle.
Branch for the posterior crico-arytenoid muscle.
Branch for the post. crico-arytænoideus.
Inferior laryngeal or recurrent nerve.

FIG. 34.
The laryngeal nerves. (After Luschka.)

events, in both cases the distance between the anterior and posterior attachments of the vocal cords is increased, and thereby the longitudinal tension of the latter is augmented. The striking of the higher notes would therefore depend materially upon the action of the crico-thyroid muscles. If this pair of muscles is paralyzed—for instance, by section of the external branch of the superior laryngeal nerve (Jelenffy, Schech)—the striking of high notes is impossible. In unilateral paralysis of the nerve in question, the vocal cord also often showed an excavation of its inner border, and lay on a different plane from that on which the cord of the non-paralyzed side lay. At the same time Henle[1] remarks, it appears to me with entire justice, that, on the one hand, the filling out of the excavation on the edge of the vocal cord cannot, on anatomical grounds, be ascribed to the crico-thyroid muscles, and that, on the other hand, it is hardly to be supposed that this muscle, even if, on a larger scale, it should give to the vocal cords that longitudinal tension corresponding to the various heights of tones, should be able to produce those fine differences in the tension of the vocal cords which determine the number of their vibrations, so much the less as the loose crico-thyroid articulation is far too coarse an apparatus to serve as a basis for such exceedingly delicate processes as the formation of the voice in singing. For the finer shades of tension of the vocal cords, as well as for transforming the bow-shaped margin into a straight line, it is evident that the internal thyro-arytenoid muscle, situated within the vocal cord, is indispensable.

The **inferior** or **recurrent laryngeal nerve,** the principal motor nerve of the laryngeal muscles, first leaves the pneumogastric—as we all know—at a variable height within the cavity of the chest, to wit, on the left side, at the height of the concavity of the aortic arch, around which it passes backwards and upwards; on the right side, at the height of the right subclavian artery, around which it also passes backwards; and then, passing the apex of the lung, runs upwards and inwards, approaching the trachea. Both recurrent nerves run at first behind the lateral lobes of the thyroid gland—that is, in the furrow between trachea and œsophagus, on both sides up to the larynx. The relations of the recurrent laryngeal nerves to their surroundings in the neck, especially to the lobes of the thyroid gland, the trachea, and the œsophagus, are very plainly to be seen on the accompanying picture of Braune's transverse section (Fig. 35). Later they pass in behind the crico-thyroid articulation, at the outer border of the plate of the cricoid cartilage, giving several motor twigs direct to the posterior crico-arytenoid muscle, and sending sensitive branches through this muscle to the mucous membrane. The external branch passes upwards to the remaining muscles (the crico-arytænoidei laterales, the thyro-arytænoideus internus and externus, the thyro-epiglottideus and arytæno-epiglottideus (?)).

The functions of the muscles innervated by the recurrent laryngeal nerve are very various, and they are in part directly antagonistic to one another.

The pair of muscles most important to life are the posterior crico-arytenoids, inasmuch as they are the only openers of the glottis. By drawing on the muscular process of the arytenoids, they rotate these cartilages upon their articular surface outwards and backwards, separate the vocal processes from one another, and open the chink of the glottis to its well-known rhomboidal form. Paralysis of the posterior crico-arytenoid muscles, their antagonists being intact (the crico-arytænoidei laterales and the thyro-arytænoidei), is followed by the drawing inwards of the vocal processes with the vocal cords, and narrowing of the glottis to a linear slit.

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[1] Handbuch der Anatomie. 1866, II. p. 258.

The lateral crico-arytenoid muscles, by their contraction, rotate the arytenoid cartilages forwards, and thus approximate the vocal processes and cords, being, therefore, true antagonists to the posterior crico-arytenoids.

The internal thyro-arytænoidei muscles are likewise antagonists to those last named, as they draw the vocal processes forwards and downwards, and at the same time towards the median line and against one another. They probably also produce those finer changes of tension in the vocal cords which are so essential to the voice in singing, but which, at least in the higher octaves, cannot be brought about without the assistance of the coarser longitudinal stretching by the crico-thyroid muscle, as a basis.

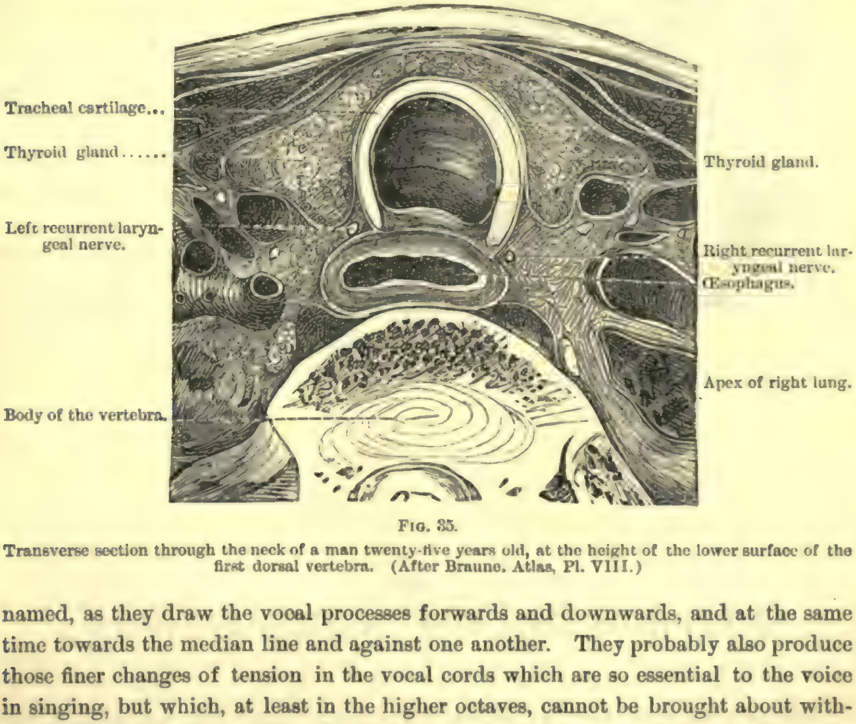

FIG. 35.

Transverse section through the neck of a man twenty-five years old, at the height of the lower surface of the first dorsal vertebra. (After Braune. Atlas, Pl. VIII.)

So far as the *sensitive function* of the recurrent laryngeal is concerned, it supplies the mucous membrane of the trachea and the lower laryngeal cavity as far as the edge of the vocal cords.

Total paralysis of conduction of the recurrent nerve would therefore make itself known by anæsthesia of the mucous membrane of the lower laryngeal cavity and paralysis of the posterior and lateral crico-arytenoid muscles, the internal and external thyro-arytenoids, and the arytænoideus (perhaps also of the thyro-epiglottideus and arytæno-epiglottideus), therefore by immobility of both vocal cords in the position in which we find them on the dead body, and which, for brevity, I have termed the *cadaveric position*, it being a medium between the positions they occupy in deepest inspiration and in phonation (compare Fig. 36).

If now, in closing, we group the muscles according to their chief functions, we shall find the following groups:

Glottis openers: the posterior crico-arytenoid muscles.

Glottis closers: the lateral crico-arytenoids, the internal and external thyro-arytenoids, and the arytænoideus.

Tensors of the vocal cords: the crico-thyroids and internal thyro-arytenoids (with the help of the posterior crico-arytenoids to fix the arytenoid cartilages posteriorly).

Depressors of the epiglottis: the thyro-epiglottideus and arytæno-epiglottideus muscles.

A sphincter of the vestibulum laryngis is constituted by the combined action of the arytæno-epiglottideus muscles, the external thyro-arytenoids and the arytænoideus muscle.

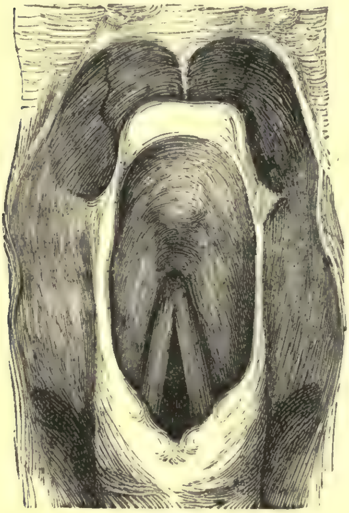

FIG. 36.

Cadaveric position of the vocal cords, drawn from a larynx recently removed from the cadaver.

NEUROSES OF SENSATION.

Over against the extensive literature and the multitude of facts which, in the course of a few years, have been brought to light on the subject of neuroses of motion, the poverty of literature on the subject of neuroses of sensation is very striking. The introduction of the laryngeal mirror for a long time remained without any influence on the study of neuroses of sensibility. It is only within the last few years that some facts have been ascertained in this direction, which represent the first beginnings of more accurate knowledge concerning disturbances of sensibility in the larynx.

Anæsthesia of the Laryngeal Mucous Membrane.

W. Leube, Diphtheritische Lähmung des Rumpfes, der Extremitäten, der Fauces und der Nervi laryngei superiores pp. Aus der Erlanger med. Klinik. D. Archiv f. klin. Med. 1869. Bd. VI. p. 266.—*Schnitzler*, Ueber Sensibilitäts-neurosen des Kehlkopfes. Wiener med. Presse. 1873. Nos. 46 and 48.—*L. Acker*, Ueber Lähmung nach Diphtherie. Aus der Erlanger med. Klinik. D. Arch. f. klin. Med. 1874. Bd. XIII. p. 416.

Diminution and final extinction of sensibility in the laryngeal mucous membrane is an ordinary symptom of approaching death, and appears most evidently and early in those affections in which carbonic acid poisoning, caused by disturbance in the interchange of gases in the lungs, introduces the final scene. Under such circumstances it extends over the entire domain of the sensitive fibres of the pneumogastric nerve.

Medicines and food which at this stage are poured into the patient flow partly through the larynx into the trachea without causing reflex closure of the laryngeal entrance or cough. Even in the bronchi, the substances that have flowed in, as well as the accumulated secretions, cause only feeble efforts at coughing or none at all. This anæsthesia of the mucous membrane of the air-passages preceding death is the expression of a deficient excitability at the centre governing the pneumogastric nerve, and may be caused temporarily to disappear under the influence of powerful stimulants to respiration and circulation.

Until recently it was sometimes asserted and sometimes denied that anæsthesia of the laryngeal mucous membrane could occur from other causes.[1]

The first conclusive observations on this subject were made by me in the Clinic at Erlangen (1869 and 1873), and were published by Prof. Leube and Dr. Acker, at that time my assistants. Another (fifth) observation, not yet published, will be given below. In these five cases of diphtheritic paralysis of the organs of the throat I was enabled to demonstrate, in part complete, and in part incomplete, anæsthesia of the laryngeal mucous membrane, combined with more or less developed motor paralysis in the domain of the superior and inferior laryngeal nerves.

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[1] Under the title of "Chronischer Katarrh des Larynx, Parese des linken wahren Stimmbandes, Anæsthesie der Mucosa Laryngis," Fieber published a case (Wiener med. Wochenschrift, 1868, No. 11) occurring in a hysterical lady, in which it was concluded that there was anæsthesia of the larynx and pharynx, from the fact that the insufflation of tannin into the larynx at first produced no reaction, and subsequently aroused powerful reaction. Such suppositions are, of course, incapable of being proved.

I also found incomplete anæsthesia in two cases of hysteria, as well as in one case of advanced bulbar paralysis (glosso-laryngeal paralysis).

After me, Chairou[1] claimed that anæsthesia of the mucous membrane of the epiglottis and the laryngeal entrance was a constant and pathognomonic symptom in the earliest stages of hysteria. But his claim loses all significance when we read that his assertion is based merely upon tests of the sensibility of the parts named by touching them with the tip of the finger (!). Careful examination of a large number of hysterical persons has further shown me that this symptom is by no means constant—in fact, not even frequent.

Of late Schnitzler[2] has published several such observations of anæsthesia of the larynx in which the anæsthesia was caused partly by hysteria and partly by diphtheria of the fauces. In one girl, besides anæsthesia, there was even hyperalgesia (anæsthesia dolorosa). Unfortunately, Schnitzler did not accompany his reports with any statement concerning the condition of the motor apparatus, which might be of great value in determining the origin of these disturbances.

With regard to the *pathogenesis* of this affection, I believe the question whether these anæsthesias are of central or peripheral origin may, for the present, be answered as follows: that those dependent on hysteria and bulbar paralysis are probably due to central, and those resulting from diphtheria are probably due to peripheral, neuropathic conditions.

The following circumstances argue favorably to my conception of the peripheral origin of the diphtheritic paralyses of sensation and motion. In my five cases the anæsthesia was associated with a paralysis of the laryngeal muscular apparatus tolerably proportionate to the degree of disturbance of sensation. Furthermore, the disturbance of innervation of the larynx always appears simultaneously with the motor paralysis of the pharyngeal and palatal muscles, which, as I formerly demonstrated on the ground of electrical reaction,[1] is altogether likely to be due to a peripheral neuropathic disturbance. The diminution or failure of reflex irritability within the larynx, which I found to be constant, seemed to me, furthermore, to be of special significance, as well as another fact which I discovered, viz., that the sensitive and motor paralysis in the larynx and fauces was developed most intensely on that side on which the diphtheritic process had exclusively or chiefly run its course. (Compare, especially, Case V., given below.) I would furthermore call attention to the circumstance, first especially noticed by Acker, that the trunk of the superior laryngeal nerve lies immediately upon the middle constrictor muscle of the pharynx, and may therefore easily be subject to the same disturbances of nutrition that affect this muscle. I would, in conclusion, specially urge that the simultaneous paralysis, to a pretty nearly equal degree, of the superior and recurrent laryngeal nerves, points to disease of the trunk of the pneumogastric itself, and might throw some light upon the circumstance which H. Weber, in his admirable work on nervous disturbances and paralysis after diphtheria,[2] brings forward as being of special significance, viz., the early fall in the frequency of the pulse, the smallness of the pulse, and the tendency to fainting-fits, which may immediately lead to death.

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[1] Études cliniques sur l'hystérie. Paris, 1870.

[2] See bibliography at head of this chapter.

The *symptoms* vary according to the degree of the anæsthesia and the presence or absence of paralysis or paresis of the muscles of the epiglottis. In complete bilateral paralysis of the upper laryngeal nerves, after diphtheria, the epiglottis is not drawn down during the act of deglutition, the laryngeal opening is not closed; food, therefore, passes into the larynx without producing sensations or reflex movements. It may then pass along without hindrance into the trachea and bronchi, and here produces painful sensations and paroxysms of coughing which expel the substances that have flowed in. Laryngoscopic inspection shows the epiglottis standing upright, without any displacement, and leaning against the root of the tongue; the mucous membrane of the larynx pale or partially reddened. Touching the parts with the point of the sound proves that the mucous membrane of the epiglottis and the upper laryngeal cavity has lost its sensibility and its reflex irritability. One may tap on the arytenoid cartilages, the false and the true vocal cords, without producing any sensation, or disposition to cough, or any action whatever in the muscular apparatus of the epiglottis or the larynx.

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[1] *von Ziemssen*, Ueber die Differenz in der Erregbarkeit gelähmter Nerven und Muskeln. Berliner klin. Wochenschrift. 1866, Nos. 43 and 46.

[2] Virchow's Archiv. 1862, Bd XXV. p. 114; and Bd. XXVIII. p. 489, 1863.

If the paralysis is incomplete, the patient is aware of being touched, and a feeble cough as well as a weak contraction of the muscular apparatus that closes the larynx are the immediate result. This difference appears, in a very striking manner, when the paralysis is complete on one side and incomplete on the other, which was the condition in my fourth case (compare Acker, l. c., p. 442), and in the one given below. In such a case, as soon as one passes, with the point of the sound—on the under surface of the epiglottis, for instance, or about the arytenoid cartilages—from the anæsthetized side to the median line and so on to the healthy side, feeble reflex movements and slight sensation appear at once when the point of the sound crosses the median line.

With reference to the *extent* of the anæsthesia, downwards, in two of my cases, which promised reliable results on account of the completeness of the anæsthesia, it extended only to the free border of the vocal cords. The mucous membrane of the lower laryngeal cavity had retained its sensibility, and indeed, as it appeared, to a normal degree. These conditions, however, may vary in other cases. Schnitzler (l. c.) reports, with regard to two cases, that the mucous membrane was without sensation deep down into the trachea. In the cases thus far observed it has been impossible to determine the extent of the anæsthesia of the superior laryngeal nerve, upwards, because there was, at the same time, anæsthesia of the mucous membrane of the pharynx and palate.

Strictly speaking, there are no *subjective* symptoms of laryngeal anæsthesia. What have been cited as such by Schnitzler, for instance, belong, properly speaking, in part to the paræsthesiæ (a feeling of dryness, burning, tickling, a feeling as of the presence of a foreign body, of emptiness, of absence of the larynx, etc.), and in part to the hyperæsthesiæ and neuralgiæ which accompany the anæsthesia. The latter seem especially liable to occur in hysterical subjects.

As a type of the clinical picture, I here give place to a fifth case of diphtheritic, sensitive, and motor paralysis of the laryngeal nerves, which I recently observed.

C. N., twenty-nine years old, a tinner's apprentice from Berlin, entered the Second Medical Clinic in Munich, on the first of February, 1876, with diphtheritic angina, which was said to have existed for eight days, but not to have caused serious annoyance until within the last few days. Examination showed a diphtheritic exudation, of the size of a finger-nail, on the right tonsil, the pharyngeal mucous membrane throughout of a dark, red color, and greatly swollen. Acute laryngeal and nasal catarrh. Fever very moderate (100½° to 102¼° Fahr. in the axilla).

During the next few days moderate swelling of the lymphatic glands at the angle of the jaw was developed on the right side. Under local treatment with a concentrated alcoholic solution of carbolic acid, and washing out with salicylic acid, the diphtheritic exudation disappeared, was developed again several times, but remained confined entirely to the ulcerated right tonsil.

In the course of the second week the tonsillar ulcer perforated the anterior palatine arch. During the third week the perforating ulcer, about the size of a sixpence, lost its diphtheritic coating, and on the 22d of February (at the beginning of the fourth week) the patient was well enough to be discharged cured.

On the 9th of March, during the sixth week from the beginning of his attack of diphtheria, the patient returned. He had been made uneasy for some time by the nasal quality of his voice, and the frequency with which things passed into his nose during swallowing. Examination showed marked anæmia, great muscular weakness, especially in the legs, without ataxia, the pulse very frequent (128), full and hard. The heart, lungs, and abdominal organs were intact. The voice was very impure and rattling. There was motor paralysis of the velum, the constrictors of the pharynx, the epiglottis, and the muscular apparatus of the larynx, it being complete on the right side, while on the left side there was only paresis. There was anæsthesia of the mucous membrane of the velum and the palatine arches, of the pharynx, the epiglottis, and the interior of the larynx; it being of a very high grade on the right side, but only moderate on the left. On the application of the electrical test, the entire muscular apparatus of the regions affected showed the *reaction of degeneration* (entire failure of the muscles to respond to the faradic current and diminished reaction to the constant current)[1] —this being complete on the right side, and incomplete on the left. In addition to this, there was paresis of accommodation. The defect in the right anterior palatine arch still existed, but no longer presented an ulcerated surface.

In the course of the next few days the disturbance of sensation and motion in the right half of the palate, the pharynx, epiglottis, and interior of the larynx increased to complete paralysis and anæsthesia; on the left the paresis was also materially increased. The voice was excessively poor in tone, and rattling. On making attempts at intonation, the velum and soft parts of the pharynx were drawn slightly towards the left. The epiglottis stood erected high and leaning somewhat backwards, so that one could see its entire inner (under) surface, and on phonating "*eh*" it was drawn slightly towards the left. The right vocal cord remained immovable in the cadaveric position; the left cord acted slightly. Touching the mucous membrane with the pointed sound on the right side of the pharynx, nowhere produced any sensation or reflex contraction; but when the right half of the epiglottis or the interior of the larynx was touched, the patient thought that he experienced a slight sensation. On the left side the sensibility was also materially diminished, but was still decidedly better than on the right. The patient avoided the entrance of fluids into the larynx by swallowing carefully.

On and after the 26th of March two subcutaneous injections of nitrate of strychnia, of $\frac{1}{14}$ of a grain each, were given.

From the end of May on, the entire picture of disease began to improve. The muscular weakness became less, the gait firmer, the voice assumed a richer tone and was less rattling; corresponding to this, during the first week in April, testing of the motility and sensibility showed decided improvement. Motility was returning everywhere. The right vocal cord again showed slight movement, the left moved more freely, but the electrical test still showed perfect lack of reaction to the induced current and hardly any reaction to a strong constant current. In spite of improved sensibility there was still a perfect lack of reflex irritability on the part of the epiglottis and the larynx.

The patient, it is true, did not wait for complete recovery, as he was anxious to return home; but when he left the Clinic, on the 15th of April, sensibility and motility in the throat were almost normal, and there was only moderate general muscular weakness.

The occurrence of anæsthesia, according to our present knowledge, is confined to cases of diphtheria, hysteria, and bulbar paralysis, and even here observations are as yet very sparse. But there can hardly be a doubt that in proportion as the attention of investigators is turned to this point the frequency of the cases observed will increase, and that, moreover, in addition to the diseases just named, laryngeal anæsthesia will be found to exist in connection with other neuroses in which the pneumogastric and especially the superior laryngeal nerves are involved.

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[1] Consult *Erb*, Vol. XI. of this Cyclopædia, p. 426, et seq.

The *duration* of laryngeal anæsthesia, in general, cannot at present be determined. In the cases of diphtheritic anæsthesia, observed by me, it was from four to six weeks.

We do not as yet possess any facts with regard to the duration of hysterical anæsthesia.

The *diagnosis* is of course impossible without accurate testing of the sensibility by means of the probe, under guidance of the laryngeal mirror. Especially is the *degree* of anæsthesia of the mucous membrane on both sides, as well as the participation of the motor tract in the paralysis, and any alterations in the mucous membrane, only to be determined in this manner. The sensitiveness to touch of the several parts is to be tested by a fine probe, as well as the comparative sensibility of the right and left sides. Sensibility to pain I test by means of the electrical current, which I localize on the individual regions through a delicate laryngeal electrode. This method of examination greatly facilitates the matter of determining the extinction of reflex irritability. As a matter of course, in case of simultaneous motor paralysis, we should at once combine herewith tests for electro-muscular irritability.

Terminations and Prognosis.—According to the cases thus far on record, the termination would always be in recovery, and the prognosis of laryngeal anæsthesia, even when associated with complete paralysis of the muscles closing the larynx, would be favorable, as hitherto all cases have recovered. And yet, with the material now before us, we cannot decide the question whether the termination would be so favorable in cases of complete paralysis of both superior laryngeal nerves, if artificial nourishment were not at the proper time substituted for voluntary deglutition, which necessarily leads to pneumonia from the entrance of food into the air-passages. Of the many deaths in diphtheritic paralysis of the pharynx, occurring in his own practice and that of others, which H. Weber cites in his work, some might, no doubt, be set down to the credit of insufficient closure of the larynx. At all events, the picture presented by a case of anæsthesia of the larynx and paralysis of the muscles which close it, as our observations have taught us, is one so calculated to make us uneasy, the condition is so threatening, that we would advise great caution in making a prognosis, not only on account of the danger from threatened pneumonia due to the presence of food in the bronchi, but also on account of the defective nutrition, which, in view of the debility of the entire organism and especially of the excessive cardiac weakness, is highly suspicious.

Therapeutics.—Some cases of hysterical laryngeal anæsthesia, and even some of diphtheritic anæsthesia, without any, or with incomplete paralysis of the muscles of the epiglottis, may end in recovery without treatment as well as without diagnosis. If the disturbance is correctly recognized, then, in case of complete paralysis, all voluntary swallowing of food or drink should at once be forbidden, and in its place nourishment should be administered through an œsophageal tube and by means of enemata of meat-juice or pancreatized meat. Care is necessary in the introduction of the œsophageal tube, otherwise its point may readily pass into the larynx, which stands wide open and is without sensation, and further down into the trachea. This mistake may be avoided by introducing the tube with the finger passed far down the throat, and by causing the patient to produce a vocal sound after the tube has descended for several inches. This, of course, implies that the muscles of the vocal cords have preserved their motility, a fact which is determined by the first examination with the laryngeal mirror. Then the production of a vocal sound is the simplest and surest method of deciding whether the tube is in the œsophagus or glottis, as in the latter instance phonation would of course be impossible.

The application of *electricity* ranks first among direct curative agents, and indeed both kinds of currents should be used alternately. As long as the special indications for one or the other kind of current are not more accurately established, it will always be advisable, in each individual case, to try both kinds, and, being guided by observation, to give the preference to that current which most quickly affects the existing disturbances for good. Direct electrization through the pharynx is here decidedly to be preferred to the percutaneous application of the current. If the double electrode[1] is introduced into both pyriform sinuses, and, by lifting the handle, the points of the arms are pressed against the anterior walls of the sinuses, they will directly touch the plica nervi laryngei on each side, which runs from the base of the arytenoid cartilage to the larger cornu of the hyoid bone, and within which the superior laryngeal nerve runs. At the same time the current will pass directly through all the soft parts of the upper laryngeal cavity. The therapeutic effect is very striking; but care must be exercised, in view of the delicacy of the organs and the superficial position of the laryngeal nerve, not to employ too strong a current. A current which will produce distinct contractions of the muscular fibres of the tongue is sufficient.

Among internal remedies, according to my experience, strychnine, in solution, used hypodermically, is the most worthy of confidence. One should begin carefully, with small doses (one-fourteenth of a grain), but in adults soon rise to one-seventh of a grain, twice a day. These doses are larger than are generally recommended, but have been given by me without any detriment and with the best results. Slight toxic manifestations appear, but they vanish at once on discontinuing the drug for a few days, and by no means contraindicate its continuance.[2]

Hyperæsthesia, Paræsthesia, and Neuralgia of the Larynx.

Ruehle, Kehlkopfkrankheiten. 1866, p. 187.—*Schnitzler*, Ueber Sensibilitätsneurosen des Kehlkopfes. Wiener med. Presse. 1873. Nos. 46 and 47, pp. 1052 and 1107.—*Tobold*, Laryngoskopie und Kehlkopfkrankheiten. 1874, p. 343.—*Mandl*, Maladies du larynx. 1872, p. 758.

Our knowledge with regard to hyperæsthesia, neuralgia, and paræsthesia of the laryngeal mucous membrane is, on the whole, very meagre.

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[1] Compare *von Ziemssen*, Electricität in der Medicin. Fourth Edition. 1872, pp. 226 and 266.

[2] Compare *Acker*, Einige Erfahrungen über subcutane Strychnininjection. Aus der Erlanger med. Klinik. D. Archiv f. klin. Medicin. Bd. XIII. 1874, p. 436.

Judging by the sparse reports to be found in literature, genuine neuralgia is very rare.

Schnitzler observed a case of this kind in a game-keeper, thirty-six years of age, who had hyperalgesia of the pharynx and larynx following an attack of angina. He was driven to the verge of suicide by intolerable pain in the throat, often associated with a feeling of its being drawn together, so that he was often obliged to draw deep inspirations in order to satisfy himself that he could still breathe. A complete cure was effected, in the course of four weeks, by pencilling the laryngeal mucous membrane with chloroform and morphine. The case is not entirely pure, inasmuch as the paroxysmal character of the pain was not pronounced; nevertheless it might still be reckoned as a neuralgia.

Hyperalgesia occurs most frequently in inflammatory and ulcerative conditions of the laryngeal mucous membrane and the remaining soft parts. The mucous membrane is excessively sensitive to every irritation, whether mechanical, chemical, or thermal. At the same time, general sensation is almost always materially altered. There are not only painful sensations, in the most varied degrees of severity, sometimes burning, with the feeling of being raw, sometimes pricking, or sensations of dryness, pressure, constriction, or darting pain in the larynx, but also paræsthesiæ—above all, a very distressing sense of tickling, of the presence of a delicate foreign body, as of a hair or the like. These troubles are often very obstinate and not unfrequently continue after the primary inflammatory or destructive process has run its course.

Not seldom, however, hyperæsthesia and paræsthesia occur without having been preceded by any other demonstrable laryngeal affection, in persons who are subject to general nervousness, to hysteria, or to hypochondriasis, especially those who suffer from seminal emissions (Mandl[1]). In such patients the same trouble is usually experienced in the pharyngeal mucous membrane. On laryngoscopic examination we find either a slight degree of chronic catarrh with granular hypertrophy, or secondary atrophy of the pharyngeal mucous membrane, or great anæmia of the laryngeal and pharyngeal mucous membrane, or, finally, no anatomical disturbance whatever. The annoying sensations experienced may, by their obstinacy and long continuance, make veritable larynx-hypochondriacs out of otherwise healthy people, but especially out of nervous persons, whose vocation requires the use of the voice (preachers, teachers). Generally, however, the morbid sensations, and the disproportionately lively complaints, are merely the outgrowth of an already existing state of hypochondriasis or hysteria, which, for some unknown reason, sets up its circus in the sensitive nerves of the pharyngeal and laryngeal mucous membrane. Patients are often misled by the pain into a belief in the existence of inflammation or ulceration —laryngeal phthisis; and are generally very happily surprised at the negative discoveries of the laryngoscopist, though they may not remain permanently convinced of their truth.

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[1] Union médicale, Paris, 1854; and Maladies du Larynx, p. 758.

While, thus, in many cases, hyperæsthesia exists as a mere disturbance of general sensation, in other rare instances it is associated with *a high degree of increased reflex irritability.* This shows itself in a *nervous*, or *spasmodic cough*, is confined almost exclusively to the female sex, and occurs in paroxyms of longer or shorter duration, often returning at a given hour of the day, or only called forth by psychical disturbances. The successive cough-explosions repeat themselves for hours and even days, with a regularity and persistency, which finally become insufferable to the listener, without the patient's appearing to be materially fatigued by the forced expirations.

Not long ago, in company with one of my colleagues in this place, I saw a hysterical lady in whom such a nervous cough had lasted uninterruptedly, day and night, for several weeks, without the occurrence even of half an hour's rest.

The sound of the cough is often peculiarly altered, most frequently becoming very deep, almost growling or humming, which, as will hereafter be shown, is due to the coarse vibrations of the arytenoid cartilages and aryteno-epiglottidean folds, which are not as fixed as they should be, on account of muscular paresis.

In nervous cough there is generally no expectoration, as Ruehle has observed, though vomiting is not uncommon. Spasmodic conditions of the muscular apparatus of the larynx and pharynx are not uncommon—thus attacks of spasm of the glottis, spasmodic interference with swallowing, and continued twitching movements of the vocal cords (Schnitzler). Manifestations of sensitive and motor irritation may, at the same time, also show themselves in the domain of other nerves, and even general convulsions may supervene.

The *course and duration* of the hyperæsthesias and hyperalgesias which arise as the result of inflammation, ulcerative destruction or a neoplasm within the larynx, depend, in general, upon the course of the primary disturbance. Still it is to be remarked that those attacks of hyperæsthesia and hyperalgesia, which are so apt to accompany chronic catarrh of the pharynx and larynx in hypochondriacs, who are at the same time public speakers, often persist most obstinately after the removal of the catarrh. Those forms, too, which arise without any primary disturbance in the larynx, often in well-marked anæmia of the laryngeal mucous membrane, and following hysteria and hypochondriasis, appear, in general, pretty obstinate, so that, from the beginning, no very favorable prognosis can be made.

In the *therapeutics* of abnormally increased irritability of the sensitive nerves of the larynx, it is to be remembered, as a general rule, that where primary disturbances exist, especially chronic catarrh, our attention must first be directed to them, because upon their removal depends the cure of the anomaly in the sensitive tract. Where, on the other hand, the hyperæsthesia appears more as an independent laryngeal neurosis, a proper regard to any general disturbances that may exist, especially of hysteria and hypochondriasis, is indispensable. Here procedures calculated to effect a general revolution in the system (cold-water treatment, sea-baths, a change of climate, etc.) usually act better than any local or medicinal treatment. In hysterical subjects narcotics are particularly worthless. Schnitzler several times observed good results in men, from pencilling the laryngeal mucous membrane with chloroform and morphine. According to my experience, bromide of potassium often acts very well, in part applied directly to the laryngeal mucous membrane and in part taken internally. We must further strongly recommend the methodical blunting of the abnormal irritability of the laryngeal mucous membrane by mechanical means (the introduction of the laryngeal probe, of a little sponge wet with an astringent solution or a solution of bromide of potassium, the mild and uniform action of which Tobold justly makes prominent). If all these means prove useless, nothing remains but to try a single, energetic cauterization of the laryngeal mucous membrane with the nitrate of silver in substance, which sometimes removes the evil for a long time.

NEUROSES OF MOTION.

I. Paralyses.

The older literature, which is of no significance, may be found cited in the work of Helfft, referred to immediately below.

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A.—Paralyses in the Domain of the Superior Laryngeal Nerve.

The consideration of paralysis of the superior laryngeal nerve is naturally linked with the discussion of anæsthesia of the laryngeal mucous membrane. According to physiological experiments (see Anatomico-Physiological Introduction), complete paralysis of conduction of the superior laryngeal nerve (section of the nerve, etc.) is followed by complete anæsthesia of the mucous membrane of the upper and middle laryngeal cavity, as well as paralysis of the crico-thyroid muscles and perhaps also of the thyro-epiglottidean and aryteno-epiglottidean muscles. Corresponding to this, there is entire loss of sensibility to the touch or to pain, in those regions of the larynx referred to and in the adjoining portions of the pharynx, as well as loss of reflex irritability (vaso-motor and trophic disturbances have not, as yet, been observed), and furthermore, owing to paralysis of the crico-thyroid muscles, a rough, deep voice, and inability to produce high tones.

With regard to participation of the *depressors of the epiglottis* in the disease, the physiological experiments made thus far do not furnish sufficient material for the determination of the question. Some clinical observations, which are given below, argue in favor of the dependence of these muscles on the superior laryngeal nerve; still they are not absolutely conclusive, and the matter certainly demands further experimental as well as clinical testing. Clinically, not only will the observation of any cases of isolated paralysis of the superior laryngeal nerve be of value, but special weight should be attached to careful examination of the muscles of the epiglottis, in complete paralysis of the recurrent laryngeal nerve of one or both sides, as well in the living as in the dead subject, inasmuch as the clinical and anatomical condition of the depressors of the epiglottis in such a case might furnish very important points for determining the question before us.

Among the few clinical observations at present before us which might be of value in studying the innervation of the muscles of the epiglottis, special significance is to be attached to Kappeler's report of the result of a resection of the trunk of the pneumogastric nerve, performed by Billroth during the extirpation of a lymphatic gland sarcoma on the right side of the neck. A piece an inch and a half long was excised from the right pneumogastric nerve, about an inch and a half above the clavicle, therefore certainly below the origin of the superior laryngeal nerve. On laryngoscopic examination, thereafter, there was found *complete paralysis of the right vocal cord* (immobility in the cadaveric position), *the muscles of the epiglottis acting normally and the sensibility of the larynx remaining intact.* (It was not found possible to arrive at a definite conclusion with regard to the capacity for action of the crico-thyroid muscles.)

To these observations may be added a number of clinical cases that have come under my notice, which entirely confirm the relations of the epiglottis, as found in Kappeler's case. In one instance of complete paralysis of the recurrent laryngeal nerve on both sides, the muscular apparatus of the epiglottis performed its functions normally; in two cases of probably complete paralysis of the superior laryngeal nerve on both sides, the muscles of the epiglottis were paralyzed, while all the muscles innervated by the recurrent laryngeal performed their functions normally.

As regards the first observations, they consist of two cases of bilateral total compression of the recurrent laryngeal nerves by cancer of the œsophagus, with complete bilateral paralysis of the vocal cords and entire aphonia, the vocal cords and arytenoid cartilages occupying the cadaveric position. *In spite of this, there was normal mobility of the epiglottis and normal closure of the larynx in swallowing.* (Compare von Ziemssen, Stimmbandlähmungen. D. Archiv f. klin. Med. 1868. IV. p. 37.)

To these may be added my cases, given in outline below, of probable complete bilateral paralysis of both superior laryngeal nerves as the result of diphtheria. Detailed accounts of these cases, observed in my Clinic, have already been made public by Prof. Leube and Dr. Acker.

Barbara D., forty-five years of age, a woman who hired herself out by the day, suffered from pharyngeal diphtheria at the same time with her children, early in March, 1869. After being sick abed for fourteen days, she returned to work, but was taken sick anew, eight days later, with difficulty of swallowing, weakness, and trembling of the extremities. The latter increased, in the course of a week, to complete paralysis, the former to entire incapacity to take nourishment, as all food swallowed passed into the trachea, and, after exciting the most tormenting paroxysms of coughing, was expelled with violence through the nose. Her voice was entirely lost (paralysis of both recurrent nerves ?), and did not return till the beginning of May. There was diplopia and indistinct vision. Nourishment was given through an œsophageal tube.

On the tenth of May she entered the Erlangen Medical Clinic. The paralysis of the muscles of the extremities, trunk, neck, and eyes was receding. There was considerable disturbance of the sensibility of the skin, the two points of the æsthesiometer being distinguishable, on the hand and fingers, only at a distance of about three and a half inches apart. Her voice was nasal but loud, somewhat rough and monotonous. The palatine arches and the pendant palate were still paralyzed. The epiglottis was paralyzed, and stood erect, high and immovable, against the base of the tongue. The movements of the vocal cords and arytenoid cartilages were somewhat sluggish, but normally extensive. There was complete anæsthesia and a lack of all reflex irritability in the mucous membrane of the epiglottis, as well as of the upper and middle laryngeal cavities as far down as the free edge of the vocal cords (passing downwards below that, the probe caused coughing and pain).

Seven weeks later the patient was entirely cured.

C. F., a farmer, forty-eight years old, went through with an attack of pharyngeal diphtheria, in the latter half of October, 1872, whereupon, during November, he was seized with general lassitude, an unsteady gait, nasal voice, and difficulty of swallowing, the latter showing itself chiefly by fluids passing the wrong way, partly into the trachea, followed by coughing-fits, partly into the nasal cavity.

At *the beginning of December* his voice was rough and rattling, with a nasal *timbre*, the movements of the vocal cords, during breathing and phonation, being entirely normal. The epiglottis stood leaning backwards against the base of the tongue, perfectly immovable. There was complete anæsthesia of the mucous membrane of the larynx, and an absence of pain and of reflex irritability on its being touched with the laryngeal sound, electrode, etc.

In both the cases here communicated there is, as one may see, a very characteristic combination of anæsthesia of the laryngeal mucous membrane down to the edges of the vocal cords, and paralysis of the muscles of the epiglottis (it being doubtful with regard to the crico-thyroid muscles); while in one case no disturbance of the function of the muscles innervated by the recurrent laryngeal nerves took place at all, in the other case, as it appears, it had occurred, but had already disappeared again. It was not possible to arrive at a definite conclusion with regard to the activity of the crico-thyroid muscles. The voice was rough, but the low degree of cultivation of the patients prevented us from settling the question whether it was possible for them to strike singing tones in the higher registers. On the whole, however, these two cases give one the impression that complete anæsthesia, entire paralysis of the epiglottis, and paralysis of the crico-thyroid muscles, constitute a group of symptoms that belong together, that are independent of paralysis in the domain of the recurrent laryngeal nerve, and that can probably only find their origin in anatomical changes of the superior laryngeal nerve.

It must remain for further observations to confirm or to rectify what is doubtful in these observations.

In an *etiological* point of view, I must place diphtheria at the head of the list, inasmuch as, since my attention has been called to this point, I have found these characteristic alterations in five cases. I also find cases that might be placed under this category in older works, especially those of H. Weber.[1]

Weber's Cases, Nos. IV., V., VI., and VII., very much resemble mine, so far as the non-laryngoscopic symptoms are concerned. In Case V., which very much resembles my case I. (compare Leube, l. c.), it is stated that "on the 26th of March, the difficulties attending swallowing were increased rather than diminished. On attempting to swallow, severe cough is excited, with expulsion of the fluids through the nose, followed by great exhaustion. Weakness of the muscles of the neck is greatly increased. On being raised, the head falls forward on to the breast," etc.

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[1] Virchow's Archiv, Bd. XXV. p. 114, 1862; and Bd. XXVIII. p. 489, 1863.

Death followed on the 28th, during a fainting-fit. Amongst other things the post-mortem showed pneumonic consolidations scattered through the lower lobe of the left lung (the result of food inhaled?).

Prognosis.—The gravity of this condition, for the life of the patient, is evidently very serious if the paralysis is developed to a high degree. The introduction of nourishment by the usual channel either has to be given up, on account of the constant escape of food into the trachea, and then rapid inanition and extreme cardiac weakness follow; or feeding by the mouth is continued, in spite of the accidents of swallowing the wrong way; and then pneumonia, as the result of the entrance of food into the air-passages, inevitably follows, with exhausting paroxysms of coughing, and often death by suffocation. In view of these results, and the comparatively long duration of diphtheritic paralysis of the larynx—according to my observations, lasting from four to six weeks—the correct diagnosis of these conditions is exceedingly important, because it leads to the immediate adoption of artificial nourishment (by means of the œsophageal tube or per rectum).

Some of the cases reported by H. Weber, Gull,[1] Maingault,[2] Monckton,[3] and others, of a fatal termination of paralysis of the palate, in which post-mortem examination showed no other changes than those of lobular pneumonia, might be referred back to paralysis of the superior laryngeal nerve, causing incomplete closure of the larynx and anæsthesia of the mucous membrane, with the results, to nutrition and the function of the lungs, which follow these conditions.

When the patient is carefully nourished, when food is prevented from entering the air-passages, and he is otherwise kept under favorable conditions, diphtheritic paralysis of the larynx seems to justify a very favorable prognosis.

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[1] Lesions of the nerves in the neck and of the cervical segments of the cord after faucial diphtheria. Lancet, 1858, Vol. II. p. 5.

[2] De la paralysie diphtheritique. Paris, 1860.

[3] Second report of the medical officer of the privy council. London, 1860. Quoted by Weber.

Cases of paralysis confined to a single function of the superior laryngeal nerve have been observed repeatedly.

Paralysis of the sensitive fibres has repeatedly been observed by Schnitzler and myself, as was shown above, in the chapter on "Anæsthesia of the Laryngeal Mucous Membrane."

Paralysis of the muscles of the epiglottis, the thyreo-epiglottidean and aryteno-epiglottidean muscles, would seem to occur frequently in paralysis of the palate and larynx, usually indeed in connection with paralysis of numerous other laryngeal muscles. Undoubtedly unilateral paresis or paralysis of the epiglottis also occurs in disturbances of the recurrent nerve of one side, as has been seen by Gerhardt, Türck, and myself; still this phenomenon is by no means a frequent, much less a constant symptom of peripheral disturbance of the recurrent laryngeal nerve, and may, therefore, probably be considered merely as an instance of anomalous innervation of the depressors of the epiglottis by the recurrent instead of by the superior laryngeal nerve.

A similar state of things seems to exist with reference to paralysis of the crico-thyroid muscles. I believe I have satisfied myself that in paralysis of the recurrent nerves these muscles are not seldom paralytic, or at least paretic; and in several cases of pure paralysis of the recurrent nerve, Türck has demonstrated post-mortem atrophy and fatty degeneration in the crico-thyroid muscle of the affected side, as well as in the muscles innervated by the paralyzed recurrent, while the superior laryngeal nerve appeared normal under the microscope. Hence it appears not improbable that the motor innervation of the crico-thyroid muscles now and then depends on the recurrent nerve.

From the experiments of Jelenffy and Schech, given above, the symptoms of paralysis of the crico-thyroid muscles might be presumed to be a rough voice in the lower registers, and inability to produce high tones, furthermore imperfect approximation of the cricoid to the thyroid cartilage. These changes seem also to occur in man, as the result of paralysis of the external branch of the superior laryngeal nerve, or of lesion of the muscle. I say "seem," for the cases found by me in literature, as, for instance, those of Mackenzie (Hoarseness, p. 44, et seq.), are, without exception, not pure enough, that is, not sufficiently free from the possibility of simultaneous implication of the thyro-arytænoideus internus, and other muscles, to serve strictly as proof. Some of the further symptoms attributed to the paralysis in question are of doubtful character, among which are the following: a difference in level between the two cords on striking high tones (the healthy cord stands higher and appears longer—Riegel); visible deficiency in vibration of the vocal cords with aphonia (Bose), excavation of the edges of the cords, depression of the centre of the cords on inspiration, and its elevation on expiration and especially on phonation; and, finally, the disappearance of the vocal processes. Mackenzie, who designates the latter three points as of special importance in the laryngoscopic diagnosis of paralysis of the crico-thyroideus, furthermore calls attention to the fact that speaking is a great effort to the patient, and that the origin of the paralysis may generally be referred back to too fatiguing and too prolonged speaking, shouting, or singing.

A more accurate delineation of this disease must be left to further, specially favorable, observations.

B.—Paralyses in the Domain of the Inferior or Recurrent Laryngeal Nerve.

Pathogenesis and Etiology.

Paralyses of the recurrent, from central causes, are tolerably rare. They imply, in part, anatomically demonstrable degeneration of the roots of the pneumogastric and spinal accessory nerves on the floor of the fourth ventricle, as they are found in disease of the medulla oblongata and the pons Varolii—for instance, in what is called bulbar paralysis; in multiple cerebrospinal sclerosis; in progressive muscular atrophy; in tabes dorsalis with eventual implication of the medulla oblongata. Such cases have been observed by Kussmaul,[1] Proust,[2] Wilks,[3] Seeligmüller,[4] Charcot,[5] Gerhardt, Schnitzler, and myself.

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[1] Ueber die fortschreitende Bulbärparalyse, etc. Volkmann's Sammlung klin. Vorträge. No. 54.

[2] Gazette des Hôpitaux. 1870. Nos. 51 and 52.

[3] Guy's Hospital Report. 3 Ser. XV. 1, 1870.

[4] Ein Fall von Lähmung des Accessorius Willisii. Archiv f. Psychiatrie u. Nervenkrankheiten. III. 2, 1872.

[5] Archives de physiol. norm. et path. II. p. 246. 1870.

I have had the opportunity of observing five cases of bilateral paralysis of the vocal cords in bulbar affections, viz., two cases of glosso-pharyngo-laryngo-labial paralysis, and three cases of "sclerose en plaques." Only in the first two cases did the paralysis become complete in time, so that the voice, which had gradually been growing more impure and weaker, finally gave place to complete aphonia, the vocal cords occupying the cadaveric position. In the three cases of sclerosis *en plaques* the paralysis was incomplete, and was localized as well in the adductors as in the tensors of the cords, so that the voice was not only without intensity and endurance but also limited in tone, and finally reduced to a monotone. With these manifestations of paralysis in the domain of the recurrent, there are then combined evidences of a similar nature in the domain of the superior laryngeal nerve, viz., partial anæsthesia, difficulty of motion and a high position of the epiglottis, paralysis of the crico-thyroideus.

We shall, doubtless, have to reckon as among the central paralyses those cases of paresis and paralysis of the vocal cords which are sometimes seen in other diseases of the brain; for instance, after apoplectic or epileptic seizures, as well as those caused by general neuroses, especially by hysteria. Instances of hysterical paresis of the tensors of the vocal cords are very frequent; those due to apoplexy and epilepsy seem to be very rare.[1]

It cannot at present be determined whether or not the rare instances of *toxic paralysis of the vocal cords*, which have been observed after poisoning with lead, arsenic, or atropine, as well as the cases of *intermittent paralysis* reported by Valleix, Gerhardt, and Levison, which are characterized by a loss of voice during several hours of the day, recurring regularly, without giving evidence of a malarial nature, are to be regarded as central paralyses of the glottis.

Paralysis of the trunk of the recurrent laryngeal nerve is not rare. Its greater frequency, as compared with paralysis of the superior laryngeal nerve, is readily explained by the long course and the exposed situation of the recurrent, which exposes it to the most manifold injuries. The difference in the length and course of the two recurrent laryngeal nerves causes a difference in the frequency with which the two are diseased. The left recurrent, which is given off by the pneumogastric deeper in the chest, is exposed to various injuries (pressure by aneurism of the arch of the aorta, by enlarged bronchial glands, etc.), against which the right recurrent is protected by its position and surroundings. On the other hand, the right recurrent, by its course along the apex of the right lung, is to a high degree exposed to the indurated thickenings, which are almost constant on the pleura in cases of chronic destructive processes in the apex of the right lung. On the whole, it may probably be assumed that, on account of its anatomical arrangement, the trunk of the left recurrent laryngeal nerve is more exposed to danger, and that there-

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[1] According to Gibb, unilateral paralysis of the glottis often occurs among the subjects of recent apoplectic hemiplegia. I have thus far only seen one case of hemiparesis of the glottis in a case of apoplectic hemiplegia several months old; but no weight can be attached to such old cases. Gibb's statement will have to be tested by recent cases of apoplexy.

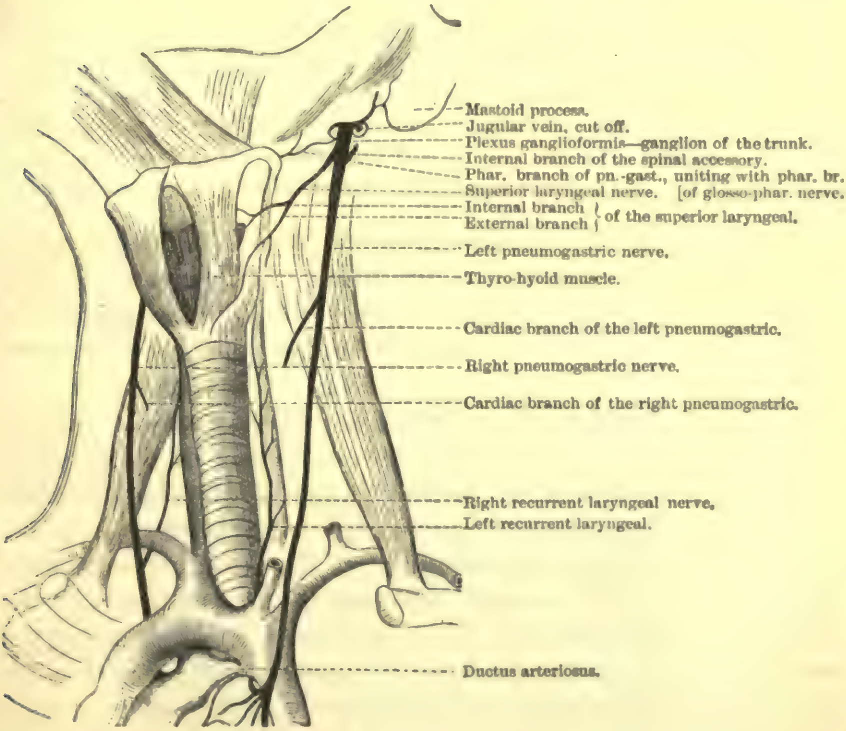
Mastoid process.
Jugular vein, cut off.
Plexus ganglioformis—ganglion of the trunk.
Internal branch of the spinal accessory.
Phar. branch of pn.-gast., uniting with phar. br. [of glosso-phar. nerve.
Superior laryngeal nerve.
Internal branch
External branch
of the superior laryngeal.
Left pneumogastric nerve.
Thyro-hyoid muscle.
Cardiac branch of the left pneumogastric.
Right pneumogastric nerve.
Cardiac branch of the right pneumogastric.
Right recurrent laryngeal nerve.
Left recurrent laryngeal.
Ductus arteriosus.

FIG. 37.

The laryngeal branches of the pneumogastric nerve in the newly born subject. (After Henle.)

fore paralysis of the recurrent is more frequent on the left side than on the right.

Among the *more rare varieties of paralysis of the fibres of the recurrent nerve* those may be mentioned first which depend on lesions of the spinal accessory within the cavity of the cranium; for instance, on tumors in the posterior cranial fossa or in the foramen lacerum. Observations of this kind have been made by Dufour,[1] Gerhardt,[2] and Türck.[3]

Türck's case of bilateral compression of the spinal accessory in both foramina lacera, through cancerous infiltration of the base of the skull, with narrowing of the foramen lacerum on both sides, dates back to pre-laryngoscopic times, and is, therefore, in spite of its significance otherwise, not to be taken as definitely proving the question before us. In this case both accessory nerves were compressed and had dwindled away. Hoarseness (without laryngeal catarrh) existed till death.

Outside of the cavity of the cranium, the first point that might be injured is the ganglion of the trunk of the pneumogastric, whereby, then, both laryngeal nerves would be paralyzed, a condition which, so far as I know, has never been encountered during life.

Below the ganglion of the trunk, after giving off the pharyngeal and the superior laryngeal nerve, the trunk of the pneumogastric may be injured by tumors in the neck or by operative procedures, being included in a ligature with the carotid, or being cut off (Kappeler); by aneurism of the carotid (Mackenzie); by gunshot or sabre wounds (Stromeyer); by carcinoma of the trunk of the nerve (Heller); by bronchocele; by aneurism of the arch of the aorta and the right subclavian artery; as well as by other tumors in the upper part of the thorax. The effect of such compression and atrophy of the trunk of the pneumogastric on the larynx will be essentially the same as that of paralysis of the recurrent laryngeal.

The disturbances which may lead to paralysis of conduction of the recurrent laryngeal nerves are pretty much the same as may affect the trunk of the pneumogastric. As a general rule, on account of its more favorable anatomical position, they act more readily and energetically on the recurrent laryngeal than on the trunk of the pneumogastric. So far as aneurisms of the arch of the aorta on the left side and of the innominate and subclavian arteries on the right side are concerned, these act on the nerves encircling them, less by pressure than by stretching them, by connective tissue adhesions and cicatricial constriction; and, indeed, according to my observations, which are now pretty numerous, these constrictions and adhesions affect, not only the recurrent, but frequently also the trunk of the pneumogastric at the same time.

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[1] Essai clinique sur le diagnostic des maladies de la voix et du larynx. Paris, 1851, p. 104. (Compression by Echinococci.)

[2] Jenaische Zeitschrift für Med. und Naturw. 1864. I. p. 485. (Carcinoma of the Base of the Skull.)

[3] Zeitschrift der k. k. Ges. der W. Aerzte. 1855, Sept. and October. Compare Klinik der Kehlkopfkrankheiten, p. 437. Wiener med. Zeitschr. No. 9, 1863.

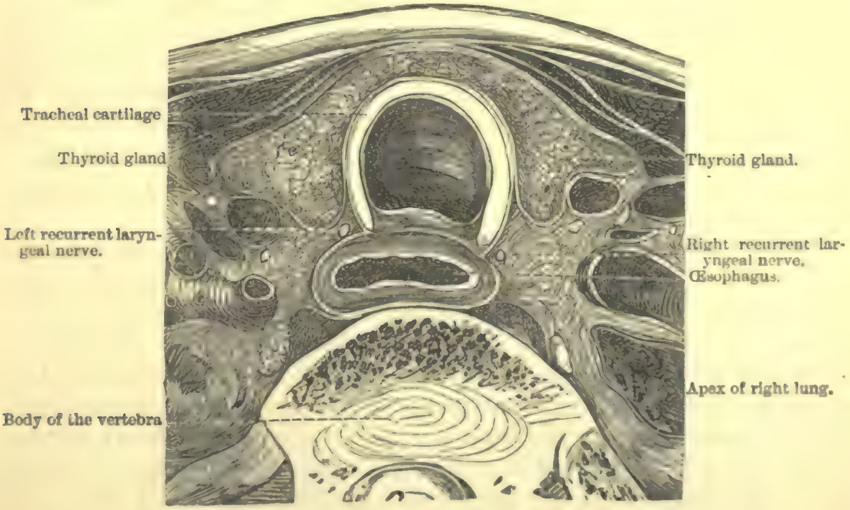

FIG. 38.

Transverse section of the neck of a man twenty five years old, at the height of the lower surface of the first dorsal vertebra. (After Braune. Atlas, Taf. VIII.)

It is self-evident that paralysis of the recurrent nerve, through the pressure of an aneurism, would usually be found only on one side—that is, on the side corresponding to the seat of the aneurism; yet the law of the duplicating of rare cases has destined me twice already to encounter bilateral paralysis of the recurrent laryngeal nerve from simultaneous aneurisms of the arch of the aorta, the innominate artery, and the subclavian artery. Traube and Munck, and Baeumler, each of them met with a case of this kind before I did.

Among the frequent causes of paralysis of the recurrent nerve, the following may be mentioned: *cancer of the œsophagus*, when situated high; *strumous hyperplasia of the thyroid gland* [goitre], especially when an acute swelling acts upon the recurrent nerve (Gerhardt), or when the tumor spreads out beneath the sternum (Türck); the development of *pleural indurations at the apex of the right* (*phthisical*) *lung*, which include the right recurrent nerve in their meshes (Mandl, Gerhardt); finally, *rheumatic influences*.

The accompanying Fig. 38 (after Braune's Atlas, Fig. VIII.; section through the lower border of the first dorsal vertebra and the top of the arch of both subclavian arteries) illustrates very prettily the relative position of both recurrent nerves to the trachea and the œsophagus, and explains, at the first glance, the frequency of the lesion of one or both recurrent nerves through cancer of the upper part of the œsophagus. Here, too, as in the case of aneurisms, it is usually not the tumor itself that surrounds and kills the nerves, but the hyperplasia and cicatricial contraction of the peri-œsophageal connective tissue. Paralysis of the vocal cords, through cancer situated high up in the œsophagus, is frequent, to judge by my tolerably numerous observations. I have seen, in all, nine cases of paralysis of the vocal cords produced in this way, four of which were bilateral. *Complete* bilateral paralysis of the vocal cords is, however, always a great rarity, here as well as in bilateral aneurism.

Braune's diagram, Fig. 38, also shows the threatening neighborhood of the *thyroid gland* to the recurrent nerve. Here it is especially the left recurrent that seems to be threatened by the left lobe of the thyroid gland. The annexed cuts, Figs. 39 and 40, which I had prepared after frozen sections of two adults—for the use of which I am indebted to the goodness of my colleague, Prof. Rüdinger—show how serious the crowding of the recurrent nerves, as well as of their neighboring organs (trachea, œsophagus, etc.), may become. In both sections the narrowing of the trachea, of the œsophagus, and of the recurrent laryngeal nerves imbedded between the two, has advanced to a very high degree. Unfortunately, nothing is known of the condition of the organs of the neck during life.

The highest grade of narrowing of the organs under consideration is represented in a section of the neck of a woman, forty-six years of age, who died of stenosis from goitre, a picture of which is given by Rüdinger in his Topographic Surgical Anatomy, 3d Division, Plate X. Fig. F. I had the opportunity of examining the original, in the possession of Prof. Rüdinger. Here the trachea is entirely separated from the œsophagus by masses of the thyroid gland, and no trace of the recurrent laryngeal nerves is to be found.

So far as a lesion of the right recurrent, by the development of chronic pleuritic indurations at the apex of the right lung, is concerned, Gerhardt estimates that there is about one instance of paralysis of the recurrent to every twelve cases of phthisis of the apex (without destructive changes in the larynx). According to my investigations, this estimate is somewhat too high. Perhaps the relative position of the recurrent nerve to the apex of the right lung is not quite the same in all men. In the accompanying drawing (Fig. 38) of Braune's (Atlas, Plate VIII.) it will be seen that the recurrent nerve lies at quite a distance from the apex of the lung. In the succeeding section by Braune (Atlas, Plate IX.), about one centimetre deeper, the nerve is found somewhat nearer to the inner margin of the upper lobe of the lung.

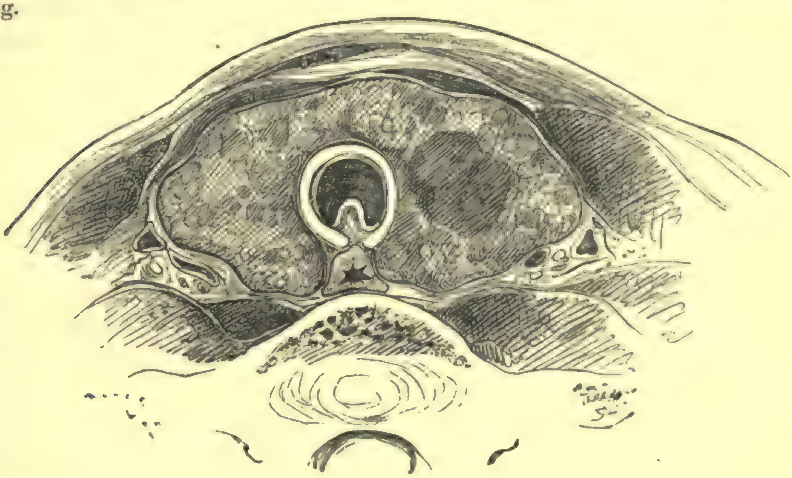

FIG. 39.

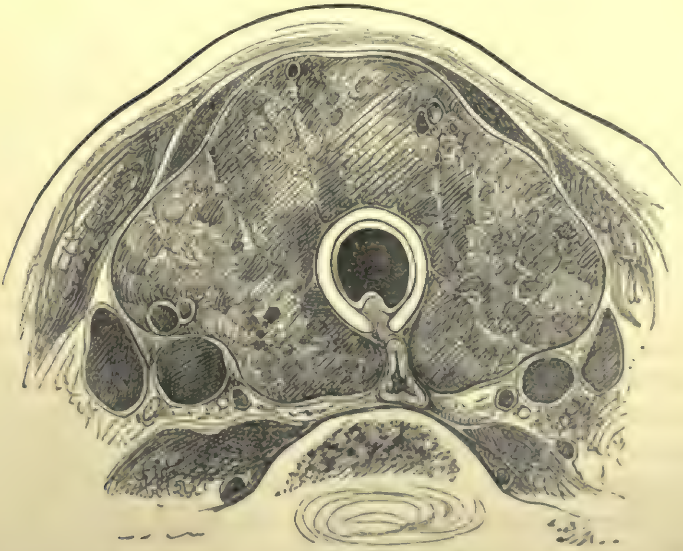

FIG. 40.

Narrowing of the trachea, the œsophagus, and both recurrent laryngeal nerves by goitre. (Drawn, after frozen sections from two different adults, by Prof. Rüdinger.)

Less frequent causes of peripheral paralysis of the recurrent laryngeal nerves are to be found in large *pericardial exudations* (Baeumler) and *tumors of the mediastinum* (cancers, sarcomas, fibromas, lymphomas of the bronchial glands: Türck, von Pastau, and others).

According to Baeumler, it is not an absolutely settled fact, and yet it is very probable, that the instance of bilateral paralysis of the vocal cords, observed by him as following a large pericardial exudation, in a debilitated syphilitic subject, depended upon paralysis of both recurrent nerves due to the crowding caused by the pericardial exudation, by the greatly enlarged heart, and by the probably over-distended right innominate and jugular veins. This appeared probable, because the loss of voice increased with the increase of the pericardial exudation, rose to the point of complete aphonia, with almost entire immobility of the vocal cords, in the cadaveric position, and gradually gave place to a normal voice again as the exudation subsided. It is a striking fact that, thus far, no further observations of the same kind have been made known. Although, since my attention was directed thereto by Baeumler's observation, I have more carefully noticed the condition of the voice and the vocal cords, in large pericardial exudations, still I have, as yet, met with no instance of paralysis of the cords.

Paralysis of individual branches of the recurrent, which go to the laryngeal muscles and to the mucous membrane of the trachea and the larynx, may arise through incomplete lesions of the trunk of the recurrent; for instance, when it is exposed to unequal pressure, or when, for any other cause, the nerve-filaments are affected, in an unequal degree, by degenerative changes. This generally takes place at the beginning of a severe lesion of a nerve, gradually leading to complete paralysis of conduction—for instance, owing to aneurism or carcinoma—and we can therefore almost always distinguish between an initial stage of incomplete paralysis—now more pronounced in one muscle, again in another—and the stage of total paralysis.

Aside from this, the individual branches of the inferior laryngeal nerve, on and in the larynx, may become paralyzed from *traumatic* causes: for instance, by gunshot, incised or punctured wounds, or pointed foreign bodies; from *excessive exertion* of the laryngeal muscles concerned; from *perichondritis*, *neoplasms*, or deep *ulcerations;* finally, from *catarrhal* and *rheumatic* influences. We must understand, however, from the start, that in the case of paralysis of individual laryngeal muscles it is very often impossible to draw a sharp line of demarcation between instances of a neuropathic and those of a myopathic origin. The subtlety of the nerves and muscles under consideration, and the fact of their being crowded together in such a small space, interposes obstacles to determining the question of the myopathic or neuropathic character of the paralysis, during life as well as on the dead subject, for the removal of which our present diagnostic aids are inadequate. Indeed, this is a question which it is often impossible to settle with reference to large muscles and motor nerves of the surface of the body—for instance, in the extremities.

Pathology.

Pathological Anatomy.

The anatomical alterations concern, on the one hand, the trunks and branches of the pneumogastric and recurrent laryngeal nerves, on the other hand, the laryngeal muscles. In the nerves we meet with the usual changes of nerves which are in process of degeneration, viz., every degree of dwindling in size, yellowish discoloration, fatty degeneration in various stages of development and of variable extent, so far as the thickness of the nerves is concerned. In peripheral paralyses of conduction, in which, during life, individual muscles alone were found paralyzed, we can, on post-mortem examination, very often demonstrate a partial degeneration confined to single nerve fasciculi. In extreme cases the recurrent nerve is found to have become as thin as paper, consisting almost exclusively of neurilemma—thus, for example, when it has become interwoven with a neoplasm.

The changes that take place in the muscles are likewise those with which we are familiar, viz., interstitial proliferation of nuclei, atrophy, and fatty metamorphosis of the primitive muscular fasciculi. In neuropathic paralysis this retrograde metamorphosis goes hand in hand with the nerve degeneration, while in myopathic cases the motor nerves, at least the *trunks* of the superior and inferior laryngeal nerves, may be found to be normal (Türck).

Symptoms and Diagnosis.

The symptoms of paralysis in the domain of the recurrent nerve are very various, according to the extent of the paralysis, according to whether it involves all or only a part of the fibres of the nerve, and whether it is unilateral or bilateral. In considering them, we will ascend from the trunk to the branches, and, in conclusion, append the study of the myopathic to that of the neuropathic paralyses.

I. Paralysis of the Trunk of the Recurrent.

a. *Bilateral Paralysis.*

Observations of bilateral paralysis of the recurrent nerve are very rare. I made public the first observation of this kind (Stimmbandlähmungen. D. Arch. f. klin. Med. 1868. Bd. IV. p. 383). This was an instance of compression of both recurrent nerves by œsophageal cancer. Aside from this, I have seen five cases of bilateral paralysis of the recurrent, two of which were caused by aneurism of the arch of the aorta and the innominate artery, and three by cancer of the œsophagus. In three of these six cases one vocal cord was not entirely though in great part paralyzed. In the same way, in a case of bilateral compression of the recurrent nerves through cancer of the thyroid gland, described by Türck (Klinik, p. 428, No. 230), the paralysis of the right vocal cord was not complete; there was also dyspnœa, probably due to compression of the trachea.

Furthermore, Traube and Munck and Baeumler have observed bilateral compression of the recurrent, which, however, was incomplete on one side, due to aneurism of the aorta and subclavian arteries, as well as the innominate of the right side. Unfortunately, no report of the autopsy is given in either of these cases. Finally, this is the right place for Baeumler's remarkable observation of bilateral incomplete paralysis of the vocal cords following extensive pericardial exudation, which was referred to under the head of Etiology.

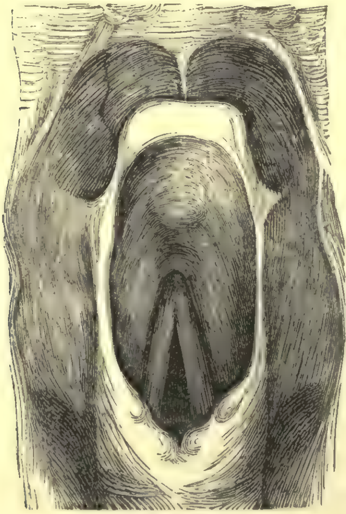

FIG. 41.

Cadaveric position of the vocal cords, drawn from a larynx removed from a recent cadaver.

As the recurrent nerve innervates all the muscles concerned in the locomotion and tension of the vocal cords (with the single exception of the crico-thyroid muscles), entire paralysis of conduction of the inferior laryngeal nerve is necessarily followed by complete immobility of the vocal cord and of the arytenoid cartilage, the former resting in the position which the cord occupies in the larynx of the dead subject. The vocal cord is of medium width, the position of its free border is half-way between the median position of phonation and the lateral position of deep inspiration. The arytenoid cartilage, both in attempts at respiration and in those at phonation, remains immovably displaced forwards and inwards. I have proposed to designate this position of the vocal cord and arytenoid cartilage as the *cadaveric position.* If both recurrent nerves are paralyzed, of course both vocal cords and arytenoid cartilages assume the cadaveric position, and then the image presented on laryngoscopic examination, as is shown in the accompanying wood-cuts (Figs. 41 and 42), corresponds perfectly with that presented by the larynx in the dead subject.

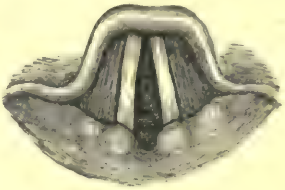

FIG. 42.

Bilateral paralysis of the recurrent nerve, caused by cancer of the œsophagus. Cadaveric position of the vocal cords. In attempts at phonation the right vocal cord still shows a trace of movement.

The symptoms of bilateral paralysis of the recurrent nerve are very striking, so that the recognition of this form of paralysis is very easy, even for an inexperienced laryngoscopist. Complete paralysis of the recurrent laryngeal nerve of both sides presents the following symptoms: absolute loss of voice; inability to cough or expectorate with force; undue expenditure of breath on making attempts at phonation, or at forcible expiration, for instance in coughing; the absence of dyspnœa during quiet breathing, at least in adults; laryngoscopically, the cadaveric position of both vocal cords, the edges of which still further approximate to each other on forced inspiration.

The absolute *aphonia* present is explained by the width of the rima glottidis, which remains unchanged in quiet respiration and in attempts at phonation, and thereby excludes the possibility of the vocal cords being thrown into vibrations by a vigorous current of air. A whispering voice is the only one possible; but even this is very fatiguing, on account of the excessive expenditure of air. Vigorous coughing and expectorating are also impossible, on account of the width of the glottis. The outlet being so large, all such attempts at forced expiration, while implying an over-exertion of the expiratory muscles, are accompanied by a powerful current of air escaping from the patient's mouth. This "phonative waste of breath"—a designation which I have proposed for this characteristic phenomenon, which is only to be observed in bilateral and not in unilateral paralysis of the vocal cords—may be sufficient, in connection with inability to cough or expectorate forcibly, as well as with extinction of the voice, to make the diagnosis pretty certain, even without laryngoscopic examination.

There is no *dyspnœa*, at least not in adults—on children no observations have as yet been made ; the patients breathe, through the sufficiently wide glottis, without the slightest inconvenience, and even the stridulous sound produced on forced inspiration is not a symptom of dyspnœa, but depends, as I have demonstrated, upon coarse vibrations produced by the strong inspiratory current of air in the soft parts of the upper laryngeal aperture (the arytenoid cartilages, which are pressed towards one another by the pressure of the air, the cartilages of Santorini, the aryteno-epiglottidean folds, and the relaxed vocal cords). The contrary assertions of Mackenzie and Evans, who claim to have observed dyspnœa in adults, I cannot regard as correct, according to my own experience, and must suppose that, in those cases, the dyspnœa depended upon other conditions which limited the space in the upper air-passage—for instance, an aneurism pressing upon the trachea. At the same time, so far as can be learned from their reports, no dyspnœa existed in the two cases of bilateral paralysis of the recurrent nerve, due to aneurisms, published by Traube and Munck, and Baeumler.

Incomplete bilateral paralysis of the recurrent nerve, whereby one vocal cord is entirely paralyzed, the other only partly so, causes the cadaveric position of one cord and a sluggish activity of the other, on phonation and coughing, an activity not quite sufficient to compensate for the paralysis of the cord on the opposite side. There is, furthermore, a deep, monotonous, impure, almost roaring voice, which can be produced only by the most active exertion of the abdominal muscles, and is accompanied with great expenditure of breath. Cough and expectoration are rendered very difficult; these acts of course demanding excessive effort on the part of the abdominal muscles, being accompanied with great expenditure of breath, and resulting in a corresponding degree of fatigue to the patient.

The grounds for the modification of this picture of incomplete as compared with complete bilateral paralysis of the vocal cords, speak for themselves. The mobility and capacity for tension, even though but slight, which are retained by one vocal cord, permits a narrowing of the glottis to a degree that limits the escape of air and throws the loosely stretched vocal cord into vibrations. The number of the vibrations depends, in each individual case, upon the degree of narrowing of the glottis, and the possibility of tension. As a matter of course, such a patient can, at first, still form a number of tones in a low register; it is but gradually that the limited tone becomes a monotone, which, with the extinction of the last remnant of muscular action, finally lapses into complete aphonia. As long as a limited tone exists, the entirely paralyzed cord may be thrown into vibrations simultaneously with the paretic cord, whereupon very striking modifications of the voice arise.

b. *Unilateral Paralysis of the Recurrent Nerve.*

The picture of the disease is composed of the following symptoms: the voice has no ring to it, and is impure (rattling), being rendered so by tremors; on straining it in speaking loud, the voice readily breaks into a falsetto, and the patient becomes wearied; the vocal cord and the arytenoid cartilage of the paralyzed side occupy the cadaveric position (see Fig. 43); during phonation the healthy vocal cord and arytenoid cartilage pass the median line, the motionless arytenoid cartilage of the other side is crowded away, whereby the glottis acquires an oblique position, and the cornicula laryngis are made to cross one another (Fig. 44); finally, unequal vibrations of the vocal cords are noticeable, being especially prominent on the production of loud and high tones.

In analyzing the above group of symptoms, their most important feature is seen to consist in *the power of the muscular apparatus of the healthy vocal cord to compensate to a certain degree the paralysis of the other cord.* This is accomplished through increased action of the adductors and tensors of the sound cord, which carry it across the median line and approximate it to the paralyzed cord, so that, on phonation, a tolerably narrow glottis is formed (which is, of course, directed obliquely towards the paralyzed side). This compensatory action of the healthy vocal cord is effected by the lateral crico-arytenoid muscle. The excessive action of this muscle turns the arytenoid cartilage so far on its axis that the vocal process passes across the median line. The arytenoid cartilage of the opposite side, no longer fixed upon its articular surface, owing to the paralysis of all its muscles, is thus crowded somewhat outwards. The arytænoideus muscle, receiving its innervation from both sides, that is, from both the right and the left recurrent nerves (and, perhaps, even from the superior laryngeal too), maintains its contractility at least in the sound half. But in view of the lack of fixation of the arytenoid cartilage, this contractility cannot show itself except in feeble twitchings, and is unable to prevent the corniculum of the sound side from passing over that of the paralyzed side. In this crossing over, the cartilage of the sound side usually passes *in front* of that of the paralyzed side (see Fig. 44), more rarely behind it.

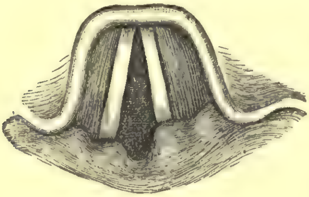
FIG. 43.
Position during inspiration in paralysis of the left vocal cord or paralysis of conduction of the left recurrent nerve.

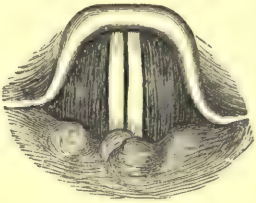
FIG. 44.
Position during phonation in paralysis of the left recurrent nerve. Crossing of the cornicula laryngis. Oblique position of the glottis.

Türck, who observed the crossing in the latter position, in one case saw even the cartilages of Wrisberg (cuneiform cartilages) cross over each other.

The usual method of crossing of Santorini's cartilages (the sound one in front) is aided by the decided sinking of the processus vocalis, which is the result of the energetic action of the lateral crico-arytænoideus and the internal thyro-arytænoideus muscles.

On forcibly striking a high tone, the healthy vocal cord may even be thrown over so as to lie upon the paralyzed cord (Kappeler).

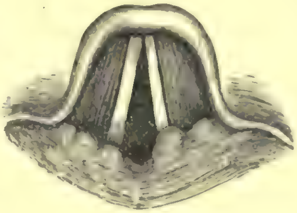

FIG. 45.
An old case of paralysis of the left recurrent nerve. Secondary atrophy of the left vocal cord.

After the existence of complete paralysis of the recurrent laryngeal nerve for years, the vocal cord becomes atrophied, and on laryngoscopic examination often—though not always—appears much narrowed, as is illustrated in Fig. 45.

That such a narrowing is not constant has been taught me again of late by the recent examination of a case of unilateral paralysis of the recurrent, which I cited in my article on paralysis of the vocal cords as Case No. VII., in which the paralysis occurred in 1859, and has therefore existed for seventeen years. In spite of the existence of complete paralysis during so many years, I here found no narrowing of the left vocal cord ; but both cords were equally wide throughout.

The absence of a normal narrowing of the glottis and of the uniform tension of both vocal cords results in a series of subjective and objective disturbances in *the formation of the voice.* In view of the abnormal width of the glottis, an excessive expenditure of breath takes place during the act of phonation, even though it be scarcely perceptible, while the air contained in the thorax is placed under heavier pressure, in order to throw the vocal cords into tone-producing vibrations, in spite of the size of the outlet. Both circumstances call the activity of abdominal pressure into play to an unusual degree, and it is therefore easy to understand why such patients should be very much exhausted by prolonged and especially loud speaking, and should experience pain at the attachment of the abdominal muscles to the walls of the thorax.

It is true that the voice becomes materially altered ; and yet, on the whole, it is less so than one would suppose. It becomes poorer in tone, impure, and altered to a higher pitch. The view held by Traube, that a falsetto voice was always developed, is not found to hold good in all cases. The paralyzed vocal cord, as a matter of course, plays a more passive rôle in the formation of the voice; being a pretty uniformly lax membrane, the expiratory current of air throws it into vibrations, which are always tolerably uniform in number. We cannot expect any material increase in the longitudinal tension of this cord from the contraction of the crico-thyroideus muscle, as there is no longer the requisite retraction of the arytenoid cartilage by the crico-arytænoideus posticus. The vicarious action of the healthy cord shows itself not only in its crossing the median line, but also in its stronger tension, as all the muscles that coöperate in the production of the voice seem to be thrown into more powerful action. The fact of the voice being pitched on a higher key might be accounted for by this more powerful tension. The frequent breaking over from a chest tone to a falsetto during loud speaking depends on vibrations of the edge of the paralyzed cord (Gerhardt), or, what seems to me more probable, of the healthy cord; or, finally, on the development of nodal points, caused by the two cords touching each other, as the result of the excessive action of the lateral crico-arytenoid muscle. The impurity of the voice, which may almost be called rattling or snarling, must be referred to the tremors that arise from the difference in the number of vibrations of the healthy and paralyzed vocal cords. The latter, on account of its laxity, will always present a far smaller number of vibrations than the healthy cord, which is, furthermore, in a state of vicarious overtension. Finally, it is self-evident that the voice must be poorer in tone than in a healthy subject, when we consider that only one cord vibrates in a chest tone, while the small number of vibrations of the paralyzed cord do not strengthen, but, on the contrary, directly disturb and vitiate the tone.

B.—Paralysis of Individual Branches of the Inferior Laryngeal Nerve and of Individual Muscles in the Domain of this Nerve.

Paralyses of individual muscles of the larynx have in many respects not yet been sufficiently studied. There is a special lack of reports of post-mortems and, above all, of microscopical examinations of the muscles and nerves of the larynx. In what follows I shall briefly treat of those points which may to a certain extent be regarded as established.

I would remark, at the outset, that I regard the division of paralysis of the laryngeal muscles into phonetic paralysis and respiratory paralysis as an unfortunate one; indeed, it is never desirable to designate a paralysis according to the *function* of the muscle which it attacks. We should regard with still less favor such an expression as "paralytic aphonia," which carries us back to the old symptomatic terminology. The object aimed at in the pathology of paralysis of the laryngeal muscles must be the same that is aimed at in that of other paralyses, viz., careful investigation of the causes, the method of occurrence, the anatomical changes, and the symptoms of paralysis *in each muscle by itself*, as has been done to a tolerably satisfactory degree for many muscles of the body. In illustration, it is only necessary to mention the valuable investigations of Duchenne into the function and disturbances of the muscles of the lower extremities. Not until the disturbances of each individual muscle have been determined will it be possible, on the basis of the special information thus won, to analyze the disturbances of a group of muscles having the same function. It will be evident, from the discussion of the subject as given hereafter, that in this endeavor we as yet encounter many obstacles.

I. Paralysis of the Posterior Crico-arytænoideus Muscle.

This pair of muscles deserve to be assigned the first place, on account of the importance of their functions in respiration and the gravity of the disturbances which follow when they are paralyzed. As was set forth above, in the anatomico-physiological introduction, the posterior crico-arytænoidei muscles constitute the only pair of muscles which have for their task the widening of the glottis essential to respiration. While this pair of muscles, by their contraction, rotate the arytenoid cartilages outwards, about their vertical axis, they at the same time separate them as far as possible from one another and draw them downwards and backwards. In this way the well-known diamond-shaped opening is produced which the glottis assumes in the deepest inspiration.

Unilateral Paralysis of the Crico-arytænoideus Posticus Muscle.

In unilateral paralysis of the crico-arytænoideus posticus muscle the contraction of its antagonists (the crico-arytænoideus lateralis, thyro-arytænoideus internus, and arytænoideus) rotates the arytenoid cartilage inwards, approximates it, as a whole, to the median line, and draws it forward, whereby the point of the vocal process is depressed. The inner border of the vocal cord of the affected side then stands almost in the median line. The glottis is wide enough for respiration, and it is only in forced inspiration that the current of air produces coarse, loud-sounding vibrations of the affected cord.

The voice is somewhat impure. Although the affected cord is still under the influence of the crico-thyroidei and thyreo-arytænoidei muscles, yet its tension cannot be raised to the normal standard on account of the lack of fixation of the arytenoid cartilage backwards. Consequently the dissimilar number of vibrations of the two cords produces tremors which make the voice sound impure and rattling to a listener, although to a far less degree than in complete unilateral paralysis of the cord. In one case, observed by me, the impurity of the voice only appeared during loud speaking, while it was hardly perceptible at other times. In one of Mackenzie's two cases, likewise, the voice is designated as slightly hoarse, while in the case of paralysis of the posterior crico-arytænoideus (lesion of the recurrent nerve through aneurism of the aorta), observed by Nicolas-Duranty, he found the voice rough and hoarse ("rauque, enrouée").

Bilateral Paralysis of the Crico-arytænoidei Postici Muscles.

The condition now under consideration is one of the gravest significance among the paralyses of individual laryngeal muscles, because upon its supervention the inspiratory opening of the glottis is extinguished, and true stenosis of the glottis of the highest grade, with danger of asphyxia, is established.

Our knowledge of this important lesion dates from within the past few years. The first observation on the subject was made public by Gerhardt, in the year 1862, in his pioneer work on Paralysis of the Vocal Cords. The number of individual observations made public since that time is still very small. After throwing out all doubtful cases (especially hysterical ones, although I consider these as belonging here), I count to-day only a total of nine cases of complete bilateral paralysis of the crico-arytænoideus posticus, as follows: one case reported by Gerhardt, two by Riegel, and one case each by Mackenzie, Pentzoldt, Feith, Nicolas-Duranty, Rehn, and myself. In spite of this paucity of material, however, the portentous picture of bilateral paralysis of the posticus muscle may be considered as well established in its most essential features.

The importance of the subject induces me to reproduce, in detail, the observations just mentioned.

I. Gerhardt's Case.

The patient was an elderly physician, who, twenty-eight years before, had contracted catarrh and shortness of breath as the result of a severe cold. (His brother is said to have suffered from inspiratory dyspnœa for years, and finally to have succumbed thereto, being highly cyanotic. Results of post-mortem, negative.) The shortness of breath persisted, and increased every winter, the inspiratory dyspnœa becoming especially severe since the application of lunar-caustic within the larynx. At the time that Gerhardt observed him the patient breathed moderately fast. every inspiration was audible, and powerful inspirations were noisy, as in a case of croup. Expiration was noiseless, easy, and short. The larynx was persistently drawn upwards (by the thyro-hyoid muscles), which made breathing easier. The same effect was produced by taking the sitting posture, with the body bent backwards and towards the left. At the same time he had a full, strongly resonant bass voice.

Laryngoscopic examination showed the epiglottis unchanged; the mucous membrane of the arytenoid cartilages somewhat swollen; the false vocal cords normal; the true cords showing fine stripes of red in some places, trembling distinctly on phonating "*eh*," *leaving a barely visible slit during inspiration*, and gaping to the width of about a line during expiration. The sensibility of the laryngeal mucous membrane was fully retained. There was frequent strangling during swallowing. The uvula and soft palate were uncommonly deeply seated. The mucous membrane of the pharynx was almost insensible to the touch.

On repeated examination of the patient, in addition to what had been found before, it was discovered that there was a moderate-sized, uniform, bilateral bronchocele. The laryngoscope, furthermore, showed a strong tendency backwards on the part of the arytenoid cartilages, as well as so strong a juxtaposition of the same that the point of the right cartilage lay somewhat before and above, principally, however, on top of, that of the left.

Electrical and other treatment alike failed to produce any result.

II. Riegel's First Case.[1]

A boy, three years of age, who, during his second year, had gone through an attack of quinsy, recovering in eight days without any bad results, was seized during his third year with an undefinable febrile affection of the chest which left him with a cough, moderate emaciation, and cyanosis on making any exertion. From the beginning of his fifth year the boy breathed somewhat more heavily. His cough was infrequent and without expectoration, his appetite good. On even slight bodily exertion there was snoring, loudly sonorous respiration, during which the patient often bent his head over backwards, stretching his neck. At the same time his voice was quite clear and resonant.

At the end of his fifth year the boy was brought to the Children's Division of the Julius Hospital. He was found to be weakly; muscular development poor; the nostrils dilating on inspiration; the lymphatic glands of the neck swollen, and one of them suppurating. The inspirations were materially lengthened and loudly sonorous; expirations short and easy. On forced inspiration there was a sinking in of the intercostal, the supra-sternal and supra-clavicular spaces, and a marked descent of the larynx. Other conditions, negative.

Laryngoscopic inspection showed the epiglottis and laryngeal mucous membrane to be pale; the glottis, even during quiet breathing, being represented only by a narrow slit, which entirely disappeared on forced inspiration, the two vocal processes touching each other. On expiration, the vocal cords separate again to the extent of the narrow slit first described. In phonation there was the normal juxtaposition and vibration of the cords, the voice being clear and pure. The sensibility of the laryngeal mucous membrane was normal.

Therapeutic interference was without result. An acute laryngitis soon made tracheotomy necessary, after which the breathing was perfectly easy.

Subsequently this patient developed a destructive pneumonia, and an attack of measles caused death.

Autopsy.—Both recurrent laryngeal nerves were compressed by tense connective tissue. The left one, lying beside the posterior portion of the aorta, between the œsophagus and trachea, was glued fast to the latter, and above that point was very much thinned. The right recurrent nerve was attached by cicatricial tissue to the right subclavian artery, two centimetres above the apex of the right lung.

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[1] Berlin klin. Wochenschrift, 1872, Nos. 20 and 21; 1873, No. 7.

The right posterior crico-arytænoideus muscle was almost entirely atrophied; the left one was not so much so. The pneumogastric and sympathetic nerves were normal. *Microscopic* examination showed atrophy of the muscular fasciculi with loss of transverse striation, more pronounced on the right side than on the left. The other laryngeal muscles were normal. The peripheral portions of both recurrent nerves showed nerve fibres that had undergone fatty degeneration and atrophy, together with others that were well preserved. There was chronic pneumonia with bronchiectasis and cheesy bronchial glands.

III. Riegel's Second Case.[1]

Towards the beginning of December, 1874, a man, fifty-eight years of age, a tourists' guide, was admitted to the Citizens' Hospital in Cologne, claiming to have been first attacked the beginning of November with dyspnœa, cough, and expectoration. There was at this time very loud and greatly prolonged inspiration, which could be heard at a great distance, while expiration was comparatively short and easy. At each inspiratory effort the larynx was much depressed. The voice was rough, but not without ring. In spite of the most extreme dyspnœa, the number of respirations was not increased. There was extensive thickening of the upper lobe of the right lung.

During respiration the vocal cords leave but a very narrow slit between them, which is still further narrowed during every deep inspiration, returning to its former calibre again during expiration. The right vocal cord is otherwise normal; the left is slightly reddened on its inner border, and has a small superficial erosion on its posterior portion. On phonation, the vocal cords come together quite to the median line, and in the succeeding inspiration separate but little.

On the very day after his admission, the dyspnœa increased to such an extent as to demand tracheotomy. The relief thereby obtained lasted but a short time, and the patient soon succumbed to his lung troubles.

Post-mortem examination revealed the following conditions, aside from the phthisical affection of the lungs and the ulcer in the mucous membrane of the left vocal cord. The posterior crico-arytænoidei muscles were of most striking, almost white sinewy appearance, showing hardly a trace of muscular tissue, while all the other laryngeal muscles seemed normal. On microscopic examination, the former showed much connective tissue lying between the muscular bundles, which were still preserved, but which showed indistinct transverse striations and granular cloudiness. The recurrent and pneumogastric nerves gave no evidences of microscopic alteration.

Accordingly the pathogenesis of the atrophy of the two crico-arytænoidei muscles remained unexplained.

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[1] *Volkmann's* Sammlung klin. Vorträge, No. 95.

IV. Pentzoldt's Case.

The subject was a woman sixty-one years of age, the widow of a day-laborer, and she gave the history of former syphilis and cerebral apoplexy. There were extensive, cicatrized, syphilitic defects of the soft palate. The glottis presented a narrow slit; the relaxed vocal cords being drawn downwards, in the form of a funnel, during inspiration. The cords were of a grayish color; the other portions of the upper division of the larynx were normal. Tracheotomy was performed on account of secondary pulmonary affection. Death followed in a few days.

Post-mortem.—The roots of the pneumogastric nerve on both sides were strikingly thin and colored gray. The spinal accessory was affected in the same way. There was distinctly gray discoloration, with a gelatinous gloss in the peripheral portions of the optic nerve; serious narrowing of the trochlear nerve; the medulla oblongata, at the level of the olivary bodies, was hard, the substance of the bodies themselves being ill defined. The anterior pyramids were distinctly gray, with a gelatinous gloss; so also were the points of origin of both pneumogastrics.

The posterior crico-arytænoidei muscles of both sides were of a pale brownish-red, shading more into a dun color than did the other muscles. The pneumogastric and recurrent laryngeal nerves of the left side were somewhat narrower and shaded into gray. Microscopically, the posterior crico-arytænoidei muscles showed indistinct transverse striation of a part of their primitive muscular fibres, a finely granular infiltration of fat, the majority of the fibres which had a distinct transverse striation containing yellow pigment. The other laryngeal muscles showed normal striation of their fibres. In the pneumogastric and the left recurrent nerves most of the primitive fibres were normal; but lying between these there were, in part normal-sized and in part distinctly widened fibres, with well-marked fatty degeneration of the neurilemma, the interstitial connective tissue being increased, with an abundant intermingling of fat cells.

V. Feith's Case.

A woman, sixty-eight years of age, was seized with acute erysipelas of the face, having, likewise, a large tumor of the spleen. On the ninth day the fever abated, and desquamation commenced. Within a few days she developed double pneumonia, to which was added difficulty of swallowing as the result of slight paresis of the soft palate. Resolution of the pneumonia began about the eleventh day, over three weeks after the commencement of the erysipelas.

At the end of the sixth week from the beginning of the attack of erysipelas, inspiratory dyspnœa was suddenly added to the difficulties of swallowing, which still existed. Within a few days this dyspnœa increased to the maximum; the voice, however, continued well preserved and pure.

Laryngoscopic examination showed that during respiration the left vocal cord remained almost in the median line, the right a little more to the outside of the median line. There was normal closure of the glottis in phonation, with a clear voice, but an entire failure of any outward movement of either vocal cord, even during deep inspiration. The vocal cords and the rest of the larynx were perfectly normal. The right vocal cord, too, was gradually moving somewhat nearer to the median line.

Two days later laryngeal dyspnœa required the performance of tracheotomy, which was followed by immediate relief. The paralysis of the soft palate was entirely cured by the application of electricity, which, however, produced no effect upon the paralysis of the posterior crico-arytenoid muscles. Subcutaneous injections of strychnine were also without any result.

Aside from the disagreeable necessity of wearing the tracheal tube, the patient thereafter enjoyed perfect comfort.

VI. Von Ziemssen's Case.

Christian Kaltenhäuser, age twenty-six, a baker's apprentice, who, with the exception of some insignificant disease of the genital organs at the age of sixteen, had always been well and very strong, was seized, without any demonstrable cause, on New Year's Day of 1871, with dyspnœa and very noisy ("howling") inspiration. At first this disturbance only appeared at night, so that he was first informed of it by his room-mates, who were disturbed during sleep by his noisy breathing. Gradually the difficulty showed itself during the day too, though at first only on his making some unusual muscular effort while at work, on his climbing stairs, on suffering from mental excitement, etc. In the meantime the stridor at night had grown so loud that his fellow-apprentices refused to sleep with him. For this reason the patient lost his situation, and after that was always soon dismissed wherever he secured employment. He claims to have been somewhat hoarse, and to have had a sense of dryness in the throat ever since his troubles began; also to have suffered somewhat from a cough, which caused extreme dyspnœa whenever the mucus was not readily loosened. At the same time he says that he never had true suffocative attacks. There was never any headache, nor other disturbance of the nervous system, nor of any other organ. His appetite was always good, and the condition of his bowels normal.

On the 11th of August, 1871, seven months and a half after the beginning of his troubles, the patient found himself compelled to take shelter in the Medical Clinic at Erlangen. The dyspnœa was moderate. There was loud stridor with every inspiration, even the most quiet; and on forced inspiration this increased to a howl that was audible in the street. Every inspiration, especially if forced, was accompanied by powerful contraction of the sterno-cleido-mastoid muscles, sinking inward of the supra-sternal and supra-clavicular region, and great descent of the larynx. The alæ of the nose remained at rest, and there was no cyanosis.

Laryngoscopic examination showed closure of the glottis, leaving but an extremely narrow slit, which was a little widened on expiration. The mucous membrane of the vocal cords and the arytenoid cartilages was feebly injected; phonation was normal, the quality of the voice being somewhat hoarse. There was slightly increased resonance on percussion at the apex of the right lung, the respiratory murmur there being, however, normal. A swelling, of the size of a goose-egg and of cartilaginous consistency, was in the right testicle and epididymis, as well as hardening of the left testicle. His pulse was small and soft. Examination in other directions resulted negatively; especially were there no traces of syphilis.

The diagnosis of paralysis of the posterior crico-arytænoidei muscles was made, and, in view of the absence of any fixed points, the nature of the paralysis was left undetermined.

After the first night the patient had to be removed to an isolated room on the ground floor, out of regard to the earnest protestations of all the other patients in his own and the neighboring wards, none of whom could sleep on account of the prodigiously loud stridor, while the patient slept soundly (as was repeatedly determined by the nurses and ourselves), and was much more noisy then than when awake. But even this room had to be vacated, for his "howls" penetrated to the floor above, disturbing the sleep of the patients in the wards over him. He was then placed alone in a barrack in the garden.

The treatment consisted in the local application of the induced and the constant current (alternately) to the paralyzed muscle, by way of the pharynx, as well as in percutaneous faradisation after the pharyngeal treatment was ended. Within four or five days there could be no question about the beneficial effect produced, and by the eighth day there was no longer any stridor when the patient was awake, while during sleep it still showed itself to a moderate degree. The mirror showed a much larger inspiratory widening of the glottis, though it was not yet normal. The movements of the right vocal cord and arytenoid cartilage were still more sluggish than those of the left.

On the 29th the difficulty seemed entirely removed, and the experiment of discontinuing the electricity was therefore tried. During the night of the 1st to the 2d of September a very severe relapse occurred; whereupon electricity was resumed on the 2d, with the best of results for the following night. This treatment was then continued regularly till the middle of the month. From that time until he left the hospital, on the 26th of September, no further difficulty was experienced, so that the patient might be considered as cured.

Unfortunately nothing could be learned of the man's further career, as he left the city.

VII. Mackenzie's Case.

An American, sixty-one years of age, claims to have had a weak voice for the past thirty years. For eighteen years he has from time to time had cramps in his throat. During the past seven or eight years he has suffered from dyspnœa, and of late, especially on making any special effort, such as climbing stairs, a loud stridulous sound accompanies it. This is so loud during sleep that it disturbs sleepers in the next room. Within the last five or six months all his difficulties have materially increased, and he has a peculiar cough and expectoration, especially in the morning. He is emaciated and of a pale, sallow, complexion.

Laryngoscopic examination shows the inner border of the vocal cords lying near the median line, the glottis not wider than one-sixteenth of an inch, enlarging, on forced expiration, to about one-eighth of an inch. On phonation, the pearly white vocal cords approximate each other. The lungs and other organs are healthy.

Tracheotomy was proposed, but was refused by the patient.

He spent the winter in Italy, took cold during the spring while crossing the Alps, and was in such danger of suffocation that on his arrival at Geneva tracheotomy had to be performed. Some months later Mackenzie found his general condition improved; the glottis, also, seemed somewhat wider during inspiration. The patient continued to wear the canula.

VIII.—Rehn's Case.

A weak, anæmic boy, thirteen years of age, who was convalescing from a severe attack of typhoid fever, which had run its course accompanied by alarming cardiac weakness, was attacked, fourteen days after the cessation of fever, with laryngeal dyspnœa on moving. This was, on the present occasion, aggravated to the highest degree by his taking a walk. During inspiration the larynx moved forcibly downwards, the supra-sternal region sank inwards, etc. His inspirations, which were long drawn,were accompanied with loud stridor, the expirations were quick and easy. There was moderate bronchitis. His voice was weak, but perfectly clear. There was great muscular weakness, especially in the legs. Tracheotomy was performed on the following day.

The laryngoscopic examination, which was now rendered possible, showed a normal mucous membrane, the glottis presenting a narrow slit, which was promptly and completely closed on attempts at phonation. On forced inspiration, there was complete linear closure of the glottis. It was impossible for the vocal cords to be moved outwards. The treatment was supporting. Strychnine produced no effect.

About eight weeks after the tracheotomy, the vocal cords showed a gradual return of their power to move outwards, the left doing better than the right.

Fifteen weeks after the operation the canula was removed. Breathing was at first still accompanied with a distinct stridor, but the patient obtained air enough, even after he had contracted an attack of acute laryngitis. He recovered slowly, but was able to walk fast and even to run some distance without trouble. Laryngoscopic exploration shows that the right vocal cord is behind the left, in point of progress.

IX.—Nicholas Duranty's Case.

Abbé X., twenty-seven years old, slender, lean, pale, had suffered for five months from aphonia, his voice having lost its power several months before. Walking caused a sense of suffocation, which was increased on climbing stairs. He was incapacitated for all intellectual effort, spent sleepless nights, had very frequent nocturnal emissions, and a capricious appetite. He had no cough; the organs of the chest were normal; no evidence of any hereditary taint. All remedies thus far applied—including hydrotherapeutics, etc.—had been without results.

Laryngoscopic examination: The laryngeal mucous membrane was slightly injected. The vocal cords, rosy in color, stood immovable, with their borders near to the median line, separated by a slit of about two mm. wide, and change their position but very little in the most varied movements of respiration.

The hyperæmia of the mucous membrane was relieved by weak solutions of nitrate of silver; but the position of the cords was in no way improved thereby, nor by the local application of electricity for a month. A residence of two months in the country improved the general nutrition; and, after this time, the vocal cords, too, appeared to move further from one another.

Nothing is known of the further course of the patient.

Aside from these nine cases, some others have been observed, which in all probability belong here, but which it is probably better to leave out of the account, because some authors are opposed to their admission to the same class as those given above. Such are Biermer's cases, Pentzoldt's Case II. (hysterical paralysis of the crico-arytænoideus posticus), and a case of paresis of this muscle by the same author,[1] then my own second case (hysterical paralysis), and finally a case of Hansen's[2] (syphilitic laryngeal affection with paralysis of the posticus muscle and almost complete cure by the inunction treatment).

So far as their bearing on **etiology** is concerned, the above cases stand as follows: Only two of them give any evidence as to the causes of this serious form of paralysis, viz., Riegel's and Pentzoldt's cases. In both of them post-mortem examinations showed it to be highly probable that they depended, essentially, upon a primary neuropathic paralysis—in one instance of the trunk of the recurrent, in the other of the trunks of the pneumogastric and spinal accessory; while in Riegel's second case the muscles were found degenerated, but the nerves intact. The remaining observations contribute but little towards elucidating the pathogenesis. In one instance the trouble began with catarrhal conditions (Gerhardt); in Rehn's case it was preceded by typhoid fever;[1] in Feith's by erysipelas with secondary pneumonia. In Mackenzie's and Nicholas-Duranty's cases, and in my own, the pathogenesis remains entirely obscure. Finally, it is very probable that hysteria may lead to transitory paralysis of these muscles.

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[1] Deutsches Archiv f. klin. Medicin. 1875, Bd. XV. p. 604.

[2] Ein Fall von isolirter Lähmung der Glottisöffner mit relativer Heilung. Petersb. med. Wochenschr. 1876, No. 6.

With regard to *age*, we find all periods represented, the patients being respectively 5, 13, 26, 27, 58, 61 (twice), and 68 years of age. Gerhardt does not give the age of his patient exactly, but he was well on in years.

As to *sex*, seven of those attacked were males and two females.

The **symptoms**, according to the record of cases thus far before us, are very striking. They consist of *the very gradual development of purely inspiratory laryngeal dyspnœa, generally without catarrh or disturbances of voice.* Moderate catarrhal manifestations and the change to a deep, rough voice constitute the exception. The signs of laryngeal stenosis on inspiration grow gradually till they reach the highest grade; so, whereas at first the inspiratory stridor only appears on making some unusual bodily exertion, such as climbing stairs, it afterwards becomes permanent, even on lying quietly in the horizontal posture, and especially during sleep. At the same time expiration usually remains quite free, and the voice loud and clear. These symptoms alone enable one to arrive at a diagnosis with a great degree of probability. Laryngoscopic examination, however, raises this to certainty. *The mirror shows the glottis transformed into a narrow slit, which narrows itself still further during inspiration;* or, speaking more correctly, it is still further narrowed by the excess of the external atmospheric pressure over that of the rarefied air within the trachea, while on expiration the glottis returns to its original size. On phonation the linear slit is narrowed in a normal manner, and the vibrations of the vocal cords show nothing abnormal.

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[1] Rehn concludes that it was myopathic paralysis as the result of typhous muscular degeneration, which had localized itself in the posterior crico-arytenoid muscles, because, in breathing, these muscles have to be unceasingly active, and are therefore, like the heart, most disposed to disintegration. This theory is certainly, to a very great degree, upset by the usual absence of paralysis of these muscles in typhoid fever; still it should stimulate us to an examination of the posterior crico-arytenoid muscles in the bodies of persons who have died of typhoid fever.

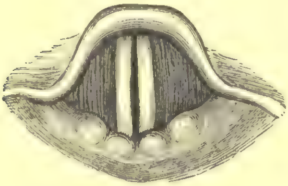

FIG. 46.

Complete bilateral paralysis of the posterior crico-arytænoidei at the moment of inspiration.

Inspiration, therefore, causes loud sounding vibrations of the vocal cords, which are pushed forward to the middle, while expiration takes place unhindered, is short and noiseless. It is, nevertheless, a singular fact that the formation of the voice so seldom undergoes any change, so far as richness and quality of tone and pitch are concerned, seeing that the posterior fixation of the arytenoid cartilages, through the posterior crico-arytenoid muscles, has failed. In the meanwhile we can hereby only be led to the conclusion that the action of these muscles is of no material consequence in the act of rendering the vocal cords tense for phonation in the median position, such as occurs in talking quietly. It still remains to be settled whether patients with paralysis of these muscles retain or lose the power of singing in the higher registers, wherein the action of these muscles might be of vital consequence.

All the other disturbances result from the narrowing of the glottis, and are the usual sequels of stenosis of the glottis and of the disturbance caused thereby to the interchange of gases in the lungs.

The *diagnosis* will but seldom present any difficulties. The conditions most likely to be confounded with this are anchylosis and the growing together of the two arytenoid cartilages. An instructive case of this kind has recently been published by Sidlo, and may here be given in brief, just because it was during life regarded as paralysis of the posterior crico-arytenoid muscles.

F. B., an infantry soldier, twenty-two years old, who had for a long time been complaining of his chest, was taken sick, seven weeks before he entered the hospital, with cough and hoarseness, to which difficulty of swallowing was soon added. Deglutition was no longer accomplished easily and with certainty; after a while pieces of food and fluids got stuck in his throat, producing a sense of suffocation, which did not cease until the article of food was expelled from the mouth or nose with violent coughing and retching. There were positively no sensations of pain.

About the same time laryngeal dyspnœa set in, especially on climbing stairs, and finally there was loud and difficult breathing day and night. The hoarseness and cough persisted, and he had no appetite.

On admission into the hospital a moderately loud, prolonged inspiratory and expiratory noise could be perceived, even at some distance. His voice was weak and hoarse. There was moderate cyanosis of the face, considerable inspiratory dyspnœa, sinking of the hollows over the sternum and clavicle, contraction of the sterno-cleido-mastoid muscles, great descent of the larynx on inspiration.

Laryngoscopic Examination.—The dark-red, greatly swelled vocal cords and false cords are in immediate contact with one another throughout the anterior third of their extent. The anterior surface of the posterior laryngeal wall, especially at the level of the vocal cords, was strongly bulged forward. Here there were some shallow excoriations as well as grayish-white indentations of the mucous membrane. The vocal cords were entirely immovable during respiration. During phonation they moved towards each other, without reaching one another in their posterior portion, where, therefore, the glottis slightly yawned.

The apex of the left lung was infiltrated, fever high, with night sweats and emaciation.

Several weeks later there was an abatement of the fever, an improvement of the general condition and of the inflammatory disturbances on the laryngeal mucous membrane. The glottis at the same time seemed even more contracted, and grew still more narrow on inspiration.

In four weeks more the glottis had closed to a barely perceptible slit, the arytenoid cartilages being in immediate contact. There was stridulous inspiration and noiseless expiration. The patient was now treated by catheterization of the glottis, and with so good a result that the glottis was brought up to half of its natural size, and he was able to breathe noiselessly during the day, even on making a moderate degree of exertion, while during sleep a somewhat louder inspiratory sound was heard, though not enough to disturb those about him.

Several months afterwards he was seized with pneumonia. Stenosis of the glottis rapidly increased to such a degree that tracheotomy had to be undertaken. Three days later he died.

Post-mortem examination showed pleuro-pneumonia and a tubercular deposit at the apex of the left lung. Aside from this, there was dislocation of both arytenoid cartilages on the anterior surface of the plate of the cricoid cartilage, their bodies lying in a horizontal position, and the two surfaces of the arytenoids facing each other being closely united by cicatricial tissue. There was a stellate cicatrix in the mucous membrane of the posterior laryngeal wall, and atrophy of both posterior crico-arytenoid muscles and of the arytænoideus muscle. The recurrent laryngeal nerves were intact.

Evidently, in this case, a deep ulcer (probably syphilitic) on the posterior wall of the larynx had healed, and the shrinking of the cicatricial connective tissue beneath and between the arytenoid cartilages had not only caused them to unite inseparably with one another, but had also dislocated them downwards and forwards. The atrophy of the posterior crico-arytenoid muscles and of the arytænoideus might be attributed to the complete cessation of their function as the result of the mechanical immobility of the arytenoid cartilages.

We see how the diagnosis in this case was rendered difficult by a very peculiar and certainly very rare combination.

The only circumstances under which there might be difficulty in distinguishing between paralysis of the posterior crico-arytenoid muscles and *cramp of their antagonists*, the adductors and tensors of the vocal cords, are these: on the one hand, when the inspiratory laryngeal dyspnœa and the characteristic laryngoscopic image are very acutely developed and rapidly reach their highest grade, and, on the other, when these disturbances are quickly evanescent, and occur in a hysterical individual, who is, otherwise, also subject to frequent cramps. The question has been raised whether stenosis of the glottis in hysterical subjects, which may last for hours or days, as has been observed by Biermer, Pentzoldt, and myself, is to be regarded as cramp or as paralysis. Basing my opinion on the observation of my own case, which is entirely similar to that of Pentzoldt, I must coincide in his opinion that the stenosis of the glottis is of paralytic and not of spastic nature. I have frequently seen spastic stenosis of the highest grade in adults, especially in hysterical subjects; but the picture presented by these spasms of the glottis is a very different thing from the one under consideration. It appears to me that, on the one hand, the persistent narrowing of the glottis for days and weeks, the absence of free intervals, the increase of the stenosis by lateral pressure, which Pentzoldt and I were able to demonstrate, the disappearance of stenosis on the occurrence of general convulsions (Pentzoldt), and the rarity of long-continued glottic cramps; and, on the other hand, the frequency of paralysis and paresis of the laryngeal muscles in hysterical subjects, all argue in favor of a transitory paralysis or paresis of the posterior crico-arytenoid muscles.

I abstain from entering any further upon this question and from introducing the observations of Biermer, Pentzoldt, and myself, because a larger amount of material seems to be necessary in order to settle the matter conclusively.

The *course* of paralysis of the crico-arytænoideus posticus has hitherto almost always been chronic. Feith's case, where the paralysis set in during convalescence from erysipelas faciei and pneumonia, is the only one in which stenosis reached such a height within a few days that tracheotomy had to be performed. In the remaining cases the difficulty usually dragged through many years. The stenosis seems to develop itself quite gradually, for the reason that the antagonists of the posticus muscles only gradually gain the ascendency and fall into a state of contraction (Schech, Riegel). In some cases the affection comes to a standstill at a moderate stage of development, so that tracheotomy does not become necessary ; in some few it may even recede (my own case, those of Hansen and Rehn, as well as hysterical paralyses). When the paralysis has existed unchanged for years, there seems to be no prospect for a cure and for removal of the tracheal canula.

Tracheotomy was necessary in six cases (Riegel's Cases 1 and 2, Pentzoldt's, Mackenzie's, Feith's, and Rehn's) ; it was not required in three, viz., in those of Gerhardt, Nicolas-Duranty, and myself—furthermore, in the hysterical paralyses seen by Pentzoldt and myself ; and, finally, in the case of paresis which Pentzoldt published. The only cures were in my own case, in those of Hansen and Rehn, and in the hysterical cases ; but even in these the patients were not long enough under observation to exclude the possibility of relapses.

Paralysis of the Crico-arytænoideus Lateralis Muscle.

The number of cases of paralysis of the lateralis muscle hitherto observed is very small. I only find them recorded by Mackenzie and Nicolas-Duranty, their reports representing both unilateral and bilateral paralysis. According to the nosographic outlines given, however, these observations leave room for the supposition that they may have been cases of total paralysis of the recurrent laryngeal nerve, or, at least, that the paralysis of the lateralis may not have been isolated. For this reason Mackenzie designates his case as paralysis of the adductors (l. c., pp. 13 and 53).

The picture presented by the disease will vary materially, as a matter of course, according as the crico-arytænoideus lateralis is affected alone or simultaneously with the rest of the antagonists of the posterior crico-arytænoideus (the thyro-arytænoidei muscles and the arytænoideus). In the latter case the glottis will be opened to the utmost extent, the vocal cords will be held firmly against the lateral walls and be incapable of any movement towards the median line.

Nicolas-Duranty's case of bilateral paralysis seems to have been of this character (l. c., p. 35). In this patient, who was a girl fifteen years old, the vocal cords stood "*immobiles sur les côtés du larynx*." The removal of the catarrhal conditions which existed at the same time made no difference in the position of the vocal cords nor in the absolute aphonia. On the other hand, a complete and permanent cure was accomplished by local treatment with electricity for four weeks.

In the case of unilateral paralysis of the lateralis which Nicolas-Duranty reports (l. c., p. 36) as occurring in a man thirty-four years old, who suffered from hoarseness, the right vocal cord was immovably fixed against the side of the larynx, and no change was produced either by the respiratory act or by attempts at phonation. Treatment by electricity and by the use of a solution of tannin effected a rapid cure.

If the thyreo-arytænoidei muscles and the arytænoideus are intact, then the vocal cord will necessarily retain to a certain extent the power of moving inwards and of tension. The sum of the disturbances due to this condition, as noticed in the formation of the voice or as seen in the laryngeal mirror, cannot at present be fixed, in view of the absence of observations of unequivocal isolated paralysis of the lateralis in man and of experimental tests of this question on animals. Therefore, at present no picture of isolated paralysis of the lateral crico-arytenoid muscle can be presented, unless it is to be of purely theoretical construction.

Paralysis of the Arytænoideus Muscle.

The arytænoideus muscle, in spite of being exposed by its position to the most varied injuries—I mention only the excessively frequent catarrhal and ulcerative processes of its investing mucous membrane—is still comparatively seldom the seat of isolated paralysis. The cause of such paralysis, whether occurring alone or in connection with that of other muscles, is most frequently to be found in acute catarrh of the posterior portion of the upper laryngeal cavity, in undue exertion of the organs of speech, and, finally, in hysterical influences.

The voice is quite hoarse. Inspection reveals the fact, aside from the catarrhal and other disturbances, that both vocal cords are normally approximated to one another during phonation in the anterior two-thirds, while the cartilaginous portion of the glottis presents an open triangle, through which the air escapes in a very perceptible manner.

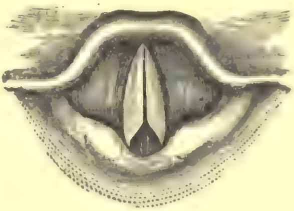

FIG. 47.

Paralysis of the arytænoideus in acute laryngitis.

In the case from which the accompanying diagram (Fig. 47) was taken, the condition was that of very severe acute catarrh of the larynx, in a beer-brewer who, while drunk, had sung loudly and shouted for a long time.

Not unfrequently the arytænoideus is subject to paralysis or paresis simultaneously with other muscles having a similar function, especially the lateral crico-arytænoidei and the thyro-arytænoidei. This probably occurs most frequently in connection with paresis of the thyro-arytænoideus internus, while the lateral crico-arytænoideus remains intact, a laryngoscopic representation of which will be given further on, under the head of Paralysis of the Internal Thyro-arytænoideus.

Paralysis of the Thyro-arytænoideus Internus Muscle, or Musculus Glottidis.

This pair of muscles seems to be affected more often than any others with paralysis and paresis. The explanation of this is easy enough. The exposed position of the muscle in the vocal cord and the great demand made upon this pair of muscles in all the more delicate processes of phonation, are enough to account for the occurrence of disease of the muscle, as well as of its motor nerves, as the result of severe and long-continued catarrh, of continuous over-exertion of the voice in prolonged speaking, singing, loud crying, and calling. Then hysteria, above all else, is liable to select this muscle as its victim, either alone or in connection with other muscles—for instance, the arytænoideus or the crico-thyreoideus. Paralysis or paresis of the internus is the most common cause of the hoarseness or aphonia so frequent in hysterical subjects.

The *symptoms* in pure cases of paralysis of the thyro-arytænoideus internus consist, as it appears, in a greater or less deficiency in tension in the vocal cords. Corresponding to this, according as the paralysis occurs on one or both sides, and is slight or serious, the disturbance varies, sometimes amounting simply to impurity of the voice, and at others to great hoarseness. Complete aphonia always implies a disturbed juxtaposition of the vocal cords and of the arytenoid cartilages—therefore, insufficient action of the laterales and the arytænoideus. The sensation of fatigue in speaking and the inability to produce loud tones are readily explained by the abnormal width of the glottis.

In the laryngoscopic image this paralysis is characterized by an excavation of the edges of the vocal cords, on phonation, which at once strikes the eye, as well as by insufficient approximation of the edges of the cords to the median line and abnormal vibrations of the same. If, as is usually the case, both internal thyro-arytenoid muscles are affected, the glottis forms a narrow oval, which extends from the tip of the vocal process to the anterior point of insertion of the vocal cords (Fig. 48).

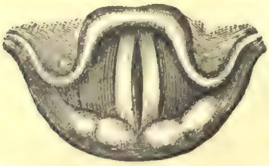

FIG. 48.
Paralysis of both thyro-arytænoidei interni muscles, as the result of acute laryngitis.

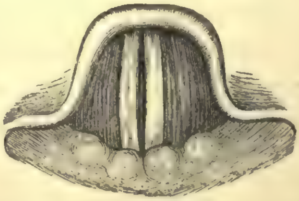

FIG. 49.
Paralysis of the thyro-arytænoideus muscle of the right side.

If the muscle of one side alone is paralyzed, the excavation is confined to the muscle of that side, as is shown in the accompanying Fig. 49.

Simultaneous paresis of the crico-thyroidei muscles, if developed to but a slight extent, will be very hard to distinguish, especially if the person is unable to sing.

The *combination of paralysis of the internus with paresis of the arytænoideus*, which is not rare, is to be recognized by a simultaneous wide yawning of the two portions of the glottis respectively included between the cords and the cartilages, while the turning inwards of the arytenoid cartilages and the consequent position of the vocal processes are almost normal. This is shown in Fig. 50.

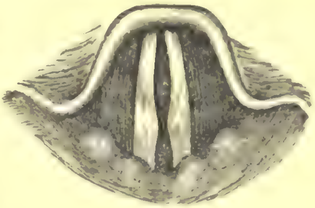

Fig. 50.

Bilateral paralysis of the thyro-arytænoideus, combined with paresis of the arytænoideus.

In such cases the formation of the voice is, of course, rendered entirely impossible by the width of the glottis.

The *course* of paralyses of the internal thyro-arytænoidei, as well as of the arytænoideus, is sometimes acute and sometimes chronic.

Paresis caused by acute laryngitis or by over-exertion of the larynx generally runs an acute course, disappearing with the abatement of the catarrh or after giving the voice a rest for several days or weeks. Those cases of paresis which have become firmly rooted through continued over-exertion of the larynx (in teachers and singers), or which depend on hysteria, are generally chronic. Still, even here a complete cure may usually be accomplished in the course of a few months, through appropriate treatment and with sufficient rest of the parts.

Diagnosis and Prognosis of Laryngeal Paralyses in General.

As a general rule, the diagnosis of laryngeal paralysis presents no difficulties to an observer of even moderate experience. It can usually be pretty definitely determined that there is paralysis and to what extent it exists. It is much more difficult and often impossible to answer the question of what causal conditions lie at the foundation of the paralysis in any individual case. This holds equally true with reference to paralysis of conduction of the trunk of the recurrent nerve and paralysis of individual muscles. In determining this question, repeated thorough examination of the organs of the neck and chest, and careful weighing of all the circumstances in the history of the case, as well as of all subjective and objective symptoms, are necessary in order to arrive at anything approaching to a positive diagnosis.

In some instances, however, not even a probable diagnosis can be made. In many of these the further progress of the case clears the matter up; sometimes, however, years of observation throw no light on the subject. I have accumulated a list of such cases, and fully agree with Schroetter when he warns his readers to be cautious about announcing the diagnosis of "rheumatic paralysis," where no cause for the paralysis can be found. Months and years often pass until, finally, an aneurism of the aorta or a carcinoma of the œsophagus comes to light as the true cause of the laryngeal paralysis.

In most instances, too, it is well to be cautious in giving a *prognosis*, not in those cases only whose pathogenesis is obscure, but also in those in which some evident and curable disturbance lies at the foundation of the paralysis, such, for instance, as a chronic laryngeal catarrh, the pressure of a goitre, an over-exertion of the organs of speech, or hysteria. While, in many cases, a cure is rapidly effected, there are those which obstinately resist the most zealous therapeutic interference without our being able to discover any reason therefor.

In hysterical paralysis of the vocal cords the result of varied therapeutic interference is at one time almost magical in its effects, at another time quite negative. The great tendency to relapse, which characterizes hysterical paralysis, must also be borne in mind in making a prognosis.

It hardly needs to be remarked that the prognosis of laryngeal paralysis, dependent on aneurism of the aorta, carcinoma of the œsophagus, a tumor in the mediastinum or at the base of the skull, or any other incurable process, is absolutely unfavorable.

Treatment.

The *treatment* of laryngeal paralysis must, first of all, be directed against the causal disturbances. In those instances of paresis, so frequently caused by laryngeal catarrh and over-exertion of the voice, a cure is generally effected by a short course of astringent and electrical local treatment, together with rest to the voice. In recent rheumatic paralysis, general diaphoretic measures, together with the Priessnitz cold-water dressing to the throat, are to be recommended. In bronchocele and swelling of the lymphatic glands in the neck and mediastinum causing compression of the recurrent laryngeal nerve, the attempt should be made to reduce their size as speedily as possible by the iodine or arsenic treatment. If anomalies in the composition of the blood or in the nervous system generally, such as chlorosis, anæmia, hysteria, and the like, seem to be the ultimate, or at least the exciting cause of the paralysis, the appropriate general treatment should be adopted. In most instances the organs of speech should be used as little as possible. But in hysterical paralysis or paresis methodical exercise of the voice—the laryngeal gymnastics of von Bruns and others—is most strongly to be recommended. This may, with advantage, be combined with mechanical irritation of the mucous membrane of the vocal cords, by the introduction of the laryngeal sound, and by the periodical lateral compression of the thyroid cartilage recommended by Olliver and Gerhardt.

Strychnine, which may be used subcutaneously to the best advantage, and in not too small doses, seems in some cases to hasten the restoration of conduction in the motor and sensitive laryngeal nerves, as well as of contraction in the muscles, as has been taught me particularly by my observations in diphtheritic paralysis (compare Acker, l. c.).

The *electrical current* is undoubtedly the most powerful and effective tonic and stimulant to paralyzed nerves and muscles, and its use is therefore indicated in the largest number of cases and in those dependent on the most varied causes. Both kinds of currents are applied in succession, in brief sessions of two or three minutes' duration, locally. They may be used *percutaneously*—that is, with both electrodes placed externally over the plates of the thyroid cartilage; or *through the pharynx*—that is, by means of a laryngeal electrode, bent like a catheter, which is intended to touch the paralyzed nerves or muscles directly.

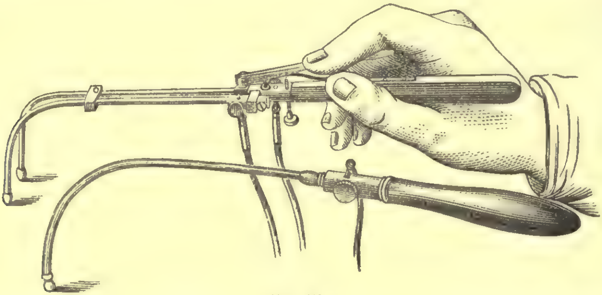

FIG. 51.

Ziemssen's single and double electrode for the pharyngeal electrization of the laryngeal muscles and nerves.

The pharyngeal localization of the electrical current is unquestionably more rational and more effective than its percutaneous application, and is therefore, in most instances, to be preferred to the latter. The single laryngeal electrode may be used, which is insulated up to its extremity, where it terminates in a little sponge-covered knob. This electrode is put in communication with one pole of the battery, while the circuit is completed by applying the other pole to the outer surface of the larynx; or the double electrode may be employed, which admits of the simultaneous introduction of both poles. With regard to the electrodes constructed at my suggestion, and represented in Fig. 51, I would merely remark that the handles are firm and compact, the arrangement of screws for the reception of the wires is strong, and that in the double electrode the two knobs can be introduced in contact with one another—and therefore inoperative—to be separated again above the laryngeal entrance by pressure on a lever. The extent of the separation of the two knobs can be regulated by an adjusting screw to be found beneath the lever.

In some cases, in which both poles are to be applied near together to the same muscle or nerve, it will be found desirable to use von Bruns' or Oertel's double electrode with immovable arms, insulated over against one another, in which the closure of the current is effected after their introduction by moving a ring or pressing upon a button.

With regard to the details of my method of localizing the current on the individual nerves and muscles of the larynx, I must refer the reader to the full explanation thereof given in my work entitled, "Die Electricität in der Medicin," 4th Edition, Berlin, 1872, pp. 226 and 262, and following.

The action of localized electricity is sometimes most brilliant, especially in hysterical paresis of tension. The aphonia, which may perhaps have lasted for months, not unfrequently disappears during the first sessions, and the normal voice takes its place. The spectacle, which strikes the layman as almost miraculous, of a hysterical patient, who for a long time has been quite voiceless, speaking only in a whisper, leaving the physician's consulting-room or electricity-room with a loud, ringing voice, is neither rare nor difficult to understand. Unfortunately the first result does not generally last long, and even if the effect of electrization always proves to be the same, yet the frequent relapses often cause the treatment to drag along for months. In simple catarrhal paresis the effect of electricity is generally rapid. The same is true of over-exertions of the voice, as well as of the paresis of the vocal cords, which often occurs at the beginning of laryngeal phthisis, before ulceration has taken place.

In the rheumatic paralyses of the recurrent nerve, and those resulting from compression, the cóndition of things is the same as in all such peripheral paralyses; the curative effect depends upon the intensity of the nerve lesion, upon the question, therefore, of whether or not there is degeneration of nerve filaments, and whether it is possible soon to restore the power of conduction. The test of the electrical irritability of the paralyzed nerves and muscles, which is of such great value in determining the prognosis of peripheral paralyses, is unfortunately not applicable here. I have only succeeded a few times, in very tolerant individuals, and those hardened to the use of electricity in the pharynx, in determining the various forms of normal, of lowered, and of degenerative reaction to the electrical current, in laryngeal paralysis, as we do in that of the palate and the pharynx.

Translator's Note.—For the information of those who do not possess a copy of von Ziemssen's admirable work on "Die Electricität in der Medicin," the text referred to on the preceding page is here given, partly in abstract, partly as a literal translation.

The electrical excitation of the laryngeal nerves and muscles has assumed a great importance in view of the facts which have been recently determined, namely, that there are frequently paralytic conditions both of the individual muscles of the larynx and also of the entire group. While many of the attempts made in recent times to excite the laryngeal branches of the vagus by applications of electricity made through the soft parts of the neck, have been followed by favorable therapeutic results, these do not necessarily show that excitation of these nerve branches has really taken place. Gerhardt attempted to ascertain the effect of such measures by observing the parts through the laryngoscopic mirror; but he reached negative or doubtful conclusions. For instance, he found that in healthy persons, when the induced current was applied to those regions of the neck which correspond to the course of the recurrents—*i. e.*, for the superior laryngeal nerves to the region of the upper cornua of the thyroid cartilage, and for the recurrents to that of the lower cornua—no visible changes were effected in the closure of the glottis or in the tension of the vocal cords, nor was any change in the vibration of the cords appreciable during the singing of notes. On the other hand, Gerhardt observed, when both sides were excited, that there was a quivering, partially successful separation of the vocal cords after pronunciation of the vowels. The glottis in one instance opened to only one-half and in another instance to three-quarters the normal width in inspiration. In unilateral excitation Gerhardt observed the same reaction only in the vocal cord of the excited side.

These manifestations are not regarded, however, by von Ziemssen as results of excitation of the recurrent, but merely as sequences to any sudden procedure upon the larynx, as in cauterization, faradisation, or galvanization, even when no special laryngeal nerves or muscles were acted upon. Successful excitation of the superior laryngeal nerve may be surely inferred, however, when the epiglottis is actively drawn down. This reaction can be obtained from within, by applying one pole of the battery to the sinus pyriformis, across which the internal branch of the superior laryngeal nerve runs obliquely, sometimes raising the mucous membrane into a fold. The author believes that the reason it so seldom happens that the inferior laryngeal nerves can be excited through the skin depends upon their deep position, covered in by layers of muscles, whose contraction resists the entrance of the electrode and the passage of the current. In these layers belong the platysma, the sterno-thyroid, sterno-hyoid, omo-hyoid, thyro-pharyngeal, and crico-pharyngeal (inferior constrictor) muscles. The recurrent nerve is very often rendered inaccessible by the enlarged lateral lobes of the thyroid gland, as well as by the fat of the neck. In extraordinary circumstances, as when these conditions do not exist, the laryngeal nerves may be reached percutaneously and good results may be obtained, especially in hysteria.

Duchenne, Mackenzie, Gerhardt, and von Bruns recognize the possibility and necessity of direct electrization from the pharynx. Tobold, Meyer, Benedikt, and others are satisfied with the percutaneous method, because of the difficulty of making internal applications and also because of the irritability of the pharyngeal and laryngeal mucous membrane. The author believes that direct electrical excitation of the laryngeal muscles can well be accomplished from the pharynx and he much prefers it; it is inapplicable, however, in the case of a single pair of muscles, which can be reached with readiness from without. This pair is the crico-thyroid muscles (musculi crico-thyreoidei recti and obliqui, Henle). These muscles, situated on the anterior portion of the neck, and stretched between the cricoid and thyroid cartilages, and on either side of the conoid ligament, are thrown into active contractions, when the electrodes are placed on each side of the conoid ligament. The fingers placed between the cartilages can then detect that they approach one another.

Excitation of individual internal laryngeal muscles from the pharynx presents at first considerable difficulties, both for the operator and for the patient. The surgeon must be familiar with the introduction of the instrument without unnecessary touching of the base of the tongue and the walls of the pharynx; he must have exact acquaintance with the anatomy of the parts, with special reference to the design of the procedure; he must be steady and sure in his application of the point of the electrode to the proper place, and, above all, he must possess a full measure of patience. In the patient, the disagreeable phenomena that at first appear, such as nausea, and indeed vomiting, active congestion of the mucous membrane, cough, transitory aphonia, etc., can only be entirely overcome after long and continuous exercises for weeks together. After this necessary perseverance on both sides, the patient's mucous membrane will at length become so tolerant to the touch of the sponge ends of the electrode and the application of the electrical excitations that the applications may be prolonged to even half a minute.

The strength of the induced or galvanic current which is to be employed, can best be determined by direct experiment upon the face. For instance, an induction current which is capable of producing distinct contractions of the frontal or corrugator muscles, or a galvanic current of from eight to ten Siemens' elements, which causes slight twitchings of the facial muscles, will amply suffice for our purposes, owing to the smallness of the laryngeal muscles, their superficial position, and the thoroughly moistened condition of the epithelial surface. The author employs as a *laryngo-electrode*, a sound shaped like a catheter and terminating in a rounded point, and fitted with a wooden handle. The point ends in a little knob, which is covered with a delicate coating of fine sponge. The double electrode (figured on page 978) is very useful for therapeutic purposes, for bilateral internal excitation, when it is desired to pass currents of any desired intensity transversely through the larynx. In studying the functions of the individual laryngeal muscles, this is not to be used, but rather the simple laryngo-electrode, or von Bruns' double electrode with very delicate branched knobs standing quite near together.

Mackenzie's laryngeal galvanizer and von Bruns' laryngo-electrode are well adapted to open or close the current at will, by pressure of the finger upon an appliance on them. When both poles are placed in the pharynx, this arrangement is specially useful. If this instrument is not at hand, then the circuit may be closed —when the laryngo-electrode has been introduced—by an assistant who has the other electrode, which is provided with a large sponge cover, and is placed either on any distant part of the body, or, in therapeutic procedures, on the surface of the larynx in an appropriate place.

The laryngo-electrode is introduced quickly with the right hand, and so as not to touch unnecessarily the walls of the passages through which it enters, while the mirror which controls the motions is held in the left hand. As for the immediate consequences of the excitations, it is impossible to avoid hyperæmia of the point excited and its neighborhood, increased secretion of mucus, hoarseness of short continuance, tendency to cough, and pain in the neck. These phenomena, however, disappear mostly after a few hours, and sometimes earlier. The therapeutic action, when such is to be expected, is seen after the disappearance of these associated phenomena, and may take place immediately or by the following morning. There is no danger that the irritation of the mucous membrane, on the posterior surface of the arytenoids, and on the sinus pyriformis, or on the vocal cords, during a long-continued course of treatment,will come to active inflammation, scabbing, or ulceration, if from time to time intervals of a few days ensue between the applications. Even when such cases have been treated continuously for several months, I have never seen local ill results.

Localization of the galvanic current upon the separate muscles may be accomplished as follows :

The **arytænoideus (transversus)** muscle is the easiest to reach with the electrode. After the apex of it has been touched on the posterior surface of the arytenoid cartilage, the circuit may be completed by an assistant employing the second electrode externally. By the shortening and thickening of the arytenoid, the posterior surface of the closely approximated cartilages will bulge forwards, and therefore special care has to be taken that when contraction ensues the point of the electrode does not slip by. The arytenoid cartilages are brought into close juxtaposition by the contraction of this muscle. Loss or incompleteness of the motion, in consequence of paralysis or paresis of the arytenoid muscle, is an important as well as frequent cause of aphonia and hoarseness. The diagnosis of this condition is not difficult even for one who has had but little experience in these matters.

The **crico-arytænoideus lateralis** is to be reached at the bottom of the sinus pyriformis, behind, in the immediate neighborhood of the external margin of the plate of the cricoid cartilage, a procedure requiring great practice and familiarity with laryngoscopic localization. It is necessary to press with the point of the electrode directly downwards and somewhat firmly, so that, by stretching the loosely spread mucous membrane, one can reach the muscle. At first there is a tendency to direct the point of the instrument in the sinus pyriformis too far forwards ; I warn against this, and advise rather to enlarge the curve of the laryngo-electrode, so as to straighten it, and to depress the handle at the moment of making the pressure.

The reaction, after exciting the crico-arytænoideus lateralis alone is slight rotation of the arytenoid on its vertical axis, turning it forwards and inwards, so that the processus vocalis, and with it the free border of the vocal cord, approaches the middle line. The glottis inter-cartilaginea will at the same time be but little moved and only so far as the cartilage, and especially its vocal process, is changed in its relation to its fellow, in the way just described. If a rapid and deep inspiration be taken while there is prolonged contraction, there will be a loud, rough purring caused by the vibrations of the projecting but lax vocal cord; at the same time the coarse vibrations of the vocal cord can be distinctly appreciated.

The **thyreo-arytænoideus externus** (Henle) or **ary-syndesmicus** (Luschka, Merkel), which adjoins immediately the anterior upper margin of the crico-thyreoideus lateralis, as well as the **thyreo-arytænoideus internus,** which lies more internally, is to be excited from the sinus pyriformis, but the position of the electrode must be so changed that the beak presses downwards, inwards, and forwards, while the handle at the same time is lifted upwards and outwards, *i. e.*, towards the upper row of teeth and the external angle of the mouth on the same side.

The effect of direct irritation of the thyreo-arytænoideus, which I obtained in this way, consists simply in bending the arytenoid cartilage forwards and downwards. Tension of the vocal cords cannot, of course, occur in this way so long as the arytenoid cartilage is held in a fixed position inwards and downwards by the arytenoid and posterior crico-arytenoid muscles, and the thyroid cartilage is approximated to the cricoid cartilage by the crico-thyroid muscles.

In these excitations in the sinus pyriformis, which require a tolerably strong pressure of the electrode, one must be careful not to mistake for muscular action the mechanical movement inwards of the very movable arytenoid cartilage with its attached true and false cords and ary-epiglottidean folds produced by the instrument. To avoid this error, one should study the position of the arytenoid cartilage and the soft parts after introducing the electrode, first, *with the circuit open*, and then *with it closed.*

It is also possible from the glottis to reach the two thyreo-arytænoidei interni, if during an inspiration the electrode is carried to or between the vocal cords. By reflex contractions of the muscles closing the glottis, the sponge-point will then be caught between the true or the false cords, and in the first case will touch the true cords at their margins, and in the latter will reach them from above. This method of excitation, however, is extremely irritating to the mucous membrane of the laryngeal entrance, and should not be repeated too often during one sitting.

The **crico-arytænoidei postici**, the important dilators of the glottis, lie upon the posterior surface of the plate of the cricoid cartilage, on both sides of a ridge passing down between them. Now, according as one is desirous of irritating the right or the left muscle, he must glide with the electrode over the posterior surface of the arytenoid, either to the right or to the left, behind the plate of the cricoid. The constrictors at first offer considerable resistance, and it is often necessary to make a vibratory motion to allow the electrode to glide down to the right place. It is also possible to pass the electrode backwards and downwards from the sinus pyriformis. As soon as the muscle contracts and becomes tumefied, the electrode slips easily to one side, for which reason the direction of the instrument is to be carefully watched.

The effect is slight rotation of the arytenoid on its axis outwards, and at the same time bending of it backwards and outwards, so that the glottis is completely opened. If, while one side is being irritated, a note is uttered, it will only be an impure and deep one, since the vocal cord of the irritated side does not advance, but only the one on the opposite side.

The **muscles of the epiglottis,** which are under the direction of the superior laryngeal nerve, namely, the **thyreo-** and **ary-epiglottici,** can either be excited by direct application on the lateral portions of the base of the epiglottis or by direct irritation of the internal branches of the superior laryngeal nerve on its way through the sinus laryngo-pharyngeus.

The **glosso-epiglotticus,** which, according to Luschka, is only the posterior one of a number of bundles from the longitudinalis linguæ superior, terminating at the epiglottis in the plica glosso-epiglottica, can scarcely ever be a matter of medical interest. At any rate, it could be reached at the Lig. glosso-epiglotticum, and more easily than could any of the other laryngeal muscles that have been mentioned.

The **vagus** is accessible for the electrode, according to some authors, from the œsophagus (Duchenne), and according to others, from the surface of the neck (Semnola, Gerhardt, Brenner). The first of these procedures presents special difficulties, because we move in darkness with the excitator. The second procedure is easier, but still of doubtful value, since, from the deep position of the vagus trunk, even when the most suitable point is selected (on the inner margin of the sterno-mastoid directly under the omo-hyoid), the thickness of the superimposed soft parts baffles usually, if not always, the irritation of the nerve. My experiments with both methods have as yet yielded me no positive results.

SPASM OF THE GLOTTIS.

STEFFEN.

SPASM OF THE GLOTTIS.

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History.

The history of spasm of the glottis is of rather recent date. The allusions which Hippocrates is said to have made to this disease, in his works, do not, with certainty, refer to it. The first writers who took pains to furnish a delineation of laryngeal cramp, and who, at the same time, asserted that one of its principal causes lay in swelling of the thymus gland, were Richa (1723) and Verdries (1726). The next works on the subject emanated from England about the year '60 of the same century. The first of these writers was James Simpson (1761), who was soon followed by Millar, Rush, and others. The work of Millar (1769) to a certain degree marked an epoch. His conception of this disease was regarded as the correct one, although, on reading his description, we find that it is but little applicable to spasm of the glottis, at most applying to cases running a very acute course, but that it refers chiefly to diseases of the respiratory organs, which ran their course with more or less serious attacks of suffocation and often terminated in death. About 1795 Wichmann, in Germany, took up the cudgels for what was called Millar's Asthma, but defined it as a form of croup without inflammation, without any material change in the mucous membrane. As a matter of course, such a conception of the nature of spasm of the glottis was not likely to contribute to any clearer understanding of the malady. Nevertheless this distorted image of Millar's asthma was clung to in Germany, although at the beginning of the present century some writers began to declare their distrust of it. In England quite able writers continued, from time to time, to associate the idea of spasm of the glottis with various diseases of the respiratory organs, with acute hydrocephalus, etc. In France but little interest had been taken in these investigations, probably on account of ignorance with regard to them. Suchet (1828) occupies the same position as Wichmann. There seem to have been no exhaustive treatises on this subject before his time. The year 1830 at once brought new life and activity into the discussion of spasm of the glottis, as Kopp published his observations, in which the attempt was made, for the first time, to give an anatomical proof of the cause of laryngeal cramp. This cause was thought to consist in hypertrophy of the thymus gland. A lively strife, pro and con, at once broke out. Various works, of various scope and weight, appeared. Those siding with Kopp were in a minority, and in some cases, too, their observations did not seem to be very accurate, as, for instance, in the eight cases which Clar, of Gratz, published in 1859.[1] The strongest argument against Kopp's views is furnished by post-mortem examinations. Bednar (1852) collected the result of thirty-nine autopsies in a table. In all of them the thymus gland was enlarged, and yet but fifteen of them, during life, had given evidence of the so-called asthma thymicum. He comes to the conclusion that the processes of disease which had taken place did not merit the name of asthma thymicum, and that the symptoms which had existed were not dependent on the enlargement of the thymus. Still more conclusive evidence against Kopp was furnished by Friedleben (1858), who established the facts with regard to the development, growth, diminution, and disappearance of the thymus gland, as well as its size, form, weight, and structure in a number of healthy cases, and furthermore threw light on the condition of this organ in various diseases. He comes to the definite conclusion that the thymus gland cannot cause asthma by pressure.

Abelin (1868) concludes, from his post-mortem investigations, that spasm of the glottis often has its origin in swelling of the thymus gland. He has also found congenital bronchocele to be a cause of this affection.

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[1] Jahrbuch für Kinderheilkunde, II. p. 106.

After the appearance of Kopp's work, opinions on the subject of spasm of the glottis were divided, chiefly in two directions. One party regards the disease as a pure idiopathic neurosis of the laryngeal nerves, and refuses to accept any other cause. The newer French school stands upon this exclusive ground, represented by such men as Valleix, Trousseau (1845), Hérard (1848), Barthez et Rilliet, Salathé (1856), Bouchut (1862), and Blache, the same views being also held by individuals in England and Germany. The opinion of the other party, that spasm of the glottis is merely a symptom of some other pathological process, and which was first advanced by Kopp, though, it is true, in a one-sided way, has given rise to the most varied anatomical explanations of the cause of this disease. Most of the adherents of this view incline to the opinion that pathological alterations in the brain are capable of producing spasm of the glottis. Among these we find some English writers, and, aside from them, more particularly Caspari (1831), Kyll (1837), Gölis (1834), and Löschner (1868). Others, for instance, Corrigan, believe they have found the cause in a material or functional change in the cervical portion of the spinal cord. Elsässer (1843), Lederer (1852), Spengler, and others have sought the cause of spasm of the glottis in rachitis, and especially in what is called craniotabes. Hugh Ley (1836), Fleischmann (1871), and others have observed serious swelling and caseation of the tracheal and bronchial glands. M. Hall and Chas. West (1865) hold that laryngeal cramp is produced only by reflex irritation, and the latter considers that it is not seldom produced by the irritation of teething. The view taken by Reid (1847) rests on the same basis, and is to the effect that the spasm may be caused by retained meconium, fæcal masses, or worms. Romberg thinks that he has observed a connection between pathological conditions of the external skin and spasm of the glottis.

Of late the majority of physicians incline to the opinion that both the general views above described are in part correct, but that the purely nervous cases of this disease are far less frequent than those which are developed secondarily, on the basis of some other pathological process.

Definition of the Disease, and Circumstances of its Occurrence.

By spasm of the glottis we mean a spasmodic disturbance of motility of the larynx, occurring in paroxysms, and consisting of a greater or less degree of narrowing or even of the momentary closure of the glottis during inspiration, together with the symptoms peculiar to this condition. The paroxysm lasts from a few seconds to a few minutes, at the outside. It depends on a spastic contraction of the muscles which, under normal circumstances, are charged with the function of narrowing the glottis—that is, the two thyro-arytenoid and two lateral crico-arytenoid muscles and the arytænoideus muscle. The abnormal activity of these muscles has its foundation in a pathological irritation of the recurrent laryngeal nerve. As narrowing or closure of the glottis may, however, be caused, not only by the contraction of these muscles, but also by diminution or cessation of the activity of the muscles that open the glottis—that is, the posterior crico-arytenoid muscles—some persons think—and this is especially true of Wunderlich—that the latter condition should also be regarded as a cause of spasm of the glottis. There is no doubt that narrowing or closure of the glottis may be brought about through paralysis, by the pressure of tumors upon the pneumogastric nerve. Then, however, if we adhere to the idea of paralysis of the posterior crico-arytenoid muscles resulting from interference with the function of the nerves belonging to them, we shall have a permanent narrowing or closure of the glottis, not an alternate increase and diminution of the same, and, least of all, a free intermission. This conception of the thing is also supported by the results obtained in animals on section of both pneumogastric nerves, viz., complete and permanent closure of the glottis. When, on the other hand, tumors exercise pressure on the pneumogastric nerve, and spasm of the glottis occurs as the result, these attacks cannot be dependent on paralysis of the nerves and the muscles to which they belong, but must result from a transitory state of irritation of these nerves, which is caused by the changing hyperæmia of the tumors. If Wunderlich supports his assertion by the observation of Hugh Ley, according to which attacks of spasm of the glottis arose after the recurrent laryngeal nerve had been included in a ligature applied to the right subclavian artery, I reply that this compression cannot have been complete; otherwise, the results of the paralysis must have been continuous. My explanation of this case, on the contrary, is as follows: that the compression was incomplete, especially at the circumference of the nerve not touched by the ligature, and that the nerve, irritated by the ligature, was temporarily thrown into a state of pathological excitement. Transitory conditions of hyperæmia of the nerve and of its surroundings may also have contributed their share to the spasm of the glottis. If there are cases in which spasm of the glottis is caused by paralysis of the muscles referred to, the first attack must necessarily lead to death by suffocation.

After advancing this doctrine of the disease under consideration, I deem it best to retain the name Spasm of the Glottis, and to drop all other designations, especially because they all contain some element inconsistent with my theory. The former terminology may be found collected in Friedreich's "Krankheiten des Larynx und der Trachea," as follows: Asthma acutum et chronicum (Millar); asthma puerorum, infantile (Boerhaave, Fr. Hoffmann); suffocatio stridula (Home); catalepsis pulmonum (Hufeland); asthma thymicum (Kopp); asthma dentientium (Pagenstecher); asthma thymo-cyanoticum (Kornmaul[1]); apnœa infantum (Rösch); laryngismus stridulus (Hugh Ley, Mason Good); cerebral croup (Pretty, John Clarke); angina stridulosa (Bretonneau); pseudocroup, etc.

Barthez and Rilliet call this disease internal convulsions; Suchet, pneumolaryngalgie; Bouchut, spasme de la glotte ou phreno-glottisme, and claims to be able to distinguish it positively from false croup, or laryngite striduleuse. Chas. West calls it simply spasm of the glottis; Hood, crowing inspiration; and Gooch, child crowing.

Spasm of the glottis has been divided by several authors into an acute and a chronic form, the name and idea of Millar's asthma being rather more attached to the former, and of asthma thymicum, according to Kopp, to the latter. I regard this division as altogether unnecessary. The attacks are always acute, and each one runs its course acutely. They are, furthermore, accompanied by the same symptoms, aside from the varying intensity shown in different individuals, or even in the same individual. The only difference in their attributes, then, would be that the acute form occurs once, or but few times, the chronic form more frequently. It still remains to be proved whether the former is fatal oftener than the latter.

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[1] Dissertation über das Asthma thymicum. Zweibrücken. 1834.

As to the circumstances under which spasm of the glottis occurs, cases may be considered under two divisions—the one embracing children up to the age of puberty, the other adults.

According to Friedreich, this spasm occurs chiefly in children from the fourth to the fourteenth month of life. Wunderlich assigns the age of from one and a half to nine years to what he calls the acute form, and that of from four to ten months, principally to the chronic form. Barthez and Rilliet have observed spasm of the glottis almost exclusively in children of the age of from three weeks to eighteen months. West has collected twenty-seven cases, twenty-one of which were in children at the age of from one and a half to two years. Steiner found, among two hundred and twenty-six children, one hundred and seventy-four in the first year of life, fifty-two in the second or third. Gerhardt declares that, as a rule, the period for this disease is from the fifth to the twenty-fourth month, and that it is very rare after dentition. Of sixty-one children with spasm of the glottis seen by Henoch, thirty-nine were between the ninth and thirtieth months, and twenty-two between the second and ninth months. He has never seen a case beyond the middle of the third year. Salathé saw twenty-four cases, four in newly-born infants, nine in those of from one to six months old, six in those from six to twelve months old, one in a child twelve years old. According to my experience, by far the majority of cases occur between the age of four months and the completion of the second year. If, however, the disease is not developed till during the second year and is disposed to be tedious, it may last for a longer or shorter period beyond the limit named. It is rare before the close of the fourth month. It is likewise but seldom observed after the close of the third year, and, if it occurs, is likely to be of less intensity than during the first years of life. I have lately seen attacks of spasm of the glottis in a boy eight years old. The disease was developed without any demonstrable cause; the attacks were few and of but moderate severity, and disappeared entirely after a few weeks.

As regards sex, all observers agree that males are oftener attacked by spasm of the glottis than females.

Hérard	saw	16	cases,	12	being	boys	and	4	girls.
Lorent	“	183	“	125	“	“	“	58	“
Steiner	“	226	“	150	“	“	“	76	“
Henoch	“	61	“	49	“	“	“	12	“
Werner	“	26	“	15	“	“	“	11	“
Hachmann	“	14	“	12	“	“	“	2	“
Pagenstecher	“	18	“	14	“	“	“	4	“
Kopp	“	10	“	9	“	“	“	1	“

The above table alone amounts to a demonstration. My experience, in twenty-five years of hospital and private practice, agrees essentially with these statements, except that I have observed the disease even less frequently in the female sex. A more recent observation of Salathé, in which he found only eleven boys in twenty-four cases, is too isolated and embraces too small numbers to throw any doubt at present upon the numerous other observations on record. We do not know why the male sex is more predisposed than the female to spasm of the glottis as well as to croupous laryngitis.

Spasm of the glottis occurs principally during the cold season of the year, and the warm air of summer seems to serve as an auxiliary in the cure of this disease, if it has previously existed. Among forty-one cases observed by Henoch, twenty-one occurred in the first four months of the year, and indeed thirteen in March. Barthez and Rilliet saw the majority of their cases in winter, and to a striking degree also in the month of March. If they were developed early in the winter they were likely to terminate as winter closed. For the same reason laryngeal cramp is more frequently found in northern than in southern latitudes.

Causes.

Cases are adduced by a number of writers which are intended to furnish evidence of hereditary predisposition to this disease. These cases, however, do not show that the parents of those affected with spasm of the glottis suffered from the disease, but only that several or most of the children of a family suffered from it. Thus Gerhardt reports nine brothers and sisters, all of whom had spasm of the glottis. J. Reid saw a family of thirteen, only one of whom escaped. Werner saw two cases each, in four families, and three children in another family seized one after the other. Romberg observed five cases in one family. Although these occurrences are not exactly rare, still they do not prove a hereditary predisposition to spasm of the glottis, but a hereditary constitution which presents a favorable soil for the development of this disease through the intervention of other causes. This hereditary constitution of the body consists, in by far the larger number of cases of spasm of the glottis, in a predisposition to rachitis. We do not, of course, mean to imply that there is congenital rachitis, as it is well known that children thus affected very seldom live long. But it is not unusual to find that the parents of children affected with laryngeal cramp themselves suffered from rachitis during childhood ; while, on the other hand, such a precedent does not seem to be necessary for the development of a tendency to rachitis in several, or in all, of the children of a family. Aside from the fact that children need not have the same bodily constitution with their parents, and may therefore show a predisposition to certain definite diseases which their parents never had, and to which they were never predisposed, the general circumstances and methods of life of a family are of the most vital influence on the bodily constitution of the children, especially during the first year of life, and, in the same measure, affect the predisposition to spasm of the glottis.

Prominent among the circumstances here alluded to are those relating to residence and food. If these are continuously unfavorable, certain diseases will be developed, consisting particularly of disturbances of nutrition, which favor the occurrence of spasm of the glottis. A house or residence is unfavorable if it is too small, too cold, or too damp, especially if the degree of dampness is subject to frequent changes; furthermore, if it has no sunshine, if it cannot be sufficiently ventilated, or is occupied by too many people. Unfavorable conditions of nourishment are caused mainly by the fact that a large number of mothers cannot or will not nourish their children at the breast. But even if mothers nurse their children, the latter hardly reach the age of a few months before they are made the victims of what might really be called the hereditary predisposition of their parents to feed them with other things, too, especially with articles, either solid or fluid, containing flour. In case the child is obliged to do without the mother's breast, it is fed with milk, which of course deteriorates in quality in proportion to the size of cities, or with its substitutes, which are also chiefly of a starchy nature. As people in good circumstances are better able to afford a roomy and healthy house, to obtain a wet-nurse for their children in case of need, and to secure better means of nourishment generally, a reason is herein found for the fact, borne out by universal experience, that spasm of the glottis, and especially the diseases predisposing thereto, occur much more frequently in the families of the poor than in those of the rich.

It is often found that children, under the unfavorable nourishment spoken of, apparently flourish well—that is, they grow stout, fat being deposited among the tissues—in other words, a certain quantity of nutritive material is laid aside, but not turned to account in the body. These children then get their teeth late and learn to walk late, their attendants being satisfied with the idea that the feet cannot support the body because it is so stout. Suddenly an attack of spasm of the glottis occurs, and, on more careful examination of the body, especially of the larger fontanelle, of the excretions, or of the epiphyses of bones, if they are made more prominent by a sudden loss of fat as the result of some disease—for instance, intestinal catarrh—one becomes aware of the fact that the child in question is suffering from rachitis. In such cases, then, one would not be able to assert that the nutrition of these children, seized with spasm of the glottis, was good and appropriate. I believe that the cases described by Salathé, in which twelve out of twenty-four children seized with spasm of the glottis were of a robust and stout habit of body, come under this category.

If rachitis is developed, as the result of a hereditary tendency, or without it, as a consequence of the above-named unfavorable methods of living, it is not merely a disease of the bony framework of the body, but a deep-seated general disturbance of nutrition, which likewise affects the soft parts, showing itself, in addition to its familiar manifestation in the bones, by a marked general relaxation of the tissues, by an abnormally increased irritability of the nervous system, by generally heightened intellectual activity, especially quickness of comprehension, by a tendency to catarrh of the respiratory and digestive organs, and by pathological changes in the excretions of the body. It is very evident that such children would be more than ordinarily disposed to spasmodic attacks. As a rule, rachitis is accompanied by œdema of the brain and effusion into the ventricles, both to a variable degree, although the intellectual activity is, in many instances, increased. On the other hand, in rachitis the walls of the chest yield more to external atmospheric pressure and are not able to expand themselves sufficiently during inspiration. The well-defined form of a rachitic chest, flattened laterally, is entirely familiar. As, under such circumstances, the respiration is superficial, its frequency must make up for its lack of depth, and the same course produces an increase in the frequency of the heart's action, which was already increased somewhat above the normal standard by the greater irritability of the nervous system. If, under these circumstances, a sudden disturbance of the uniform rhythm of respiration occurs, which would be materially contributed to by the increased nervous irritability, a damming up of the blood in the sinuses of the dura mater and the cerebral veins will take place, inasmuch as the act of inspiration has a material influence on the return flow of venous blood from the cavity of the cranium. The greater the venous hyperæmia of the brain and medulla oblongata, the more powerful is the impulse to supply the body with the requisite amount of oxygen by deep inspirations. The causes which, under such circumstances, may bring about a sudden disturbance of the respiratory rhythm are manifold. The principal ones that may be mentioned are awaking out of sleep, especially being awakened, changes of the position of the body, coughing, screaming, the swallowing of food, disturbances of mind, and abrupt changes of temperature, especially from warm to cold.

The simultaneous action of all the causes then results in a laryngeal cramp, which corresponds in intensity and duration to the conditions present.

There are at the same time some other circumstances which may favor the occurrence of spasm, provided the above causes are present. In this category we find diseases of the heart, interference with respiration by elevation of the diaphragm, owing to over-filling of the stomach, or over-distention of the intestines by fæcal masses, or serious swelling of the liver. Hood, in particular, has called attention to the latter circumstance. I am, however, not of the opinion that swelling and altered function of the liver alone are capable of producing spasm of the glottis. I find it very natural that Hood should have been able to demonstrate such a condition of the liver in the majority of his cases. One can seldom make a post-mortem examination of children in the first years of life without finding partial fatty degeneration of the liver—that is, the infiltration of fat into certain larger or smaller areas of liver cells, which are generally situated at the periphery of the organ, not extending far in depth. In rachitis this filling up with fat is apt to be more diffuse, often involving the entire liver, and at the same time causing a serious enlargement of the same. If, in such cases, spasm of the glottis supervenes, it is the rachitis that is the foundation of it and not the swelled liver. The latter can only favor the occurrence of cramp, in so far that it still further permanently impedes respiration through the crowding upward of the diaphragm. The fact of spasm of the glottis not occurring in chronic tuberculosis, especially of the lungs, which, during the age of childhood, is, as a rule, associated with serious fatty infiltration and swelling of the liver, goes to prove that neither this condition of the organ nor the results of its pathologically altered function, have anything to do, in and of themselves, with spasm of the glottis.

I have thus specifically set forth the relations of rachitis to spasm of the glottis, because in by far the larger number of cases, according to my experience at least nine-tenths, the former gives occasion to the rise of the latter. Naturally enough, spasm of the glottis does not occur in all cases of rachitis. It depends upon the degree to which this chronic disturbance of nutrition is developed, and especially upon the question whether the bony framework of the chest is involved, and to what extent. A factor of equal importance is the degree of irritability of the nervous system. If these two conditions are wanting, or exist to but a limited degree, then we need not expect the occurrence of laryngeal cramp.

Elsässer regarded the rachitic softening of the occiput as the cause of spasm; others have attributed it to a hyperæmic condition of the bones in question. As craniotabes only occurs in decided, and pretty generally developed rachitis, spasm of the glottis may be expected when it is present, but in no way depends on it, and does not necessarily follow. The theory that the paroxysms are called forth by pressure on the back of the head, has long since been overthrown, inasmuch as children are attacked by it not only while lying down, but also when sitting up.

Loeschner, who has striven in the most praiseworthy manner to find the cause of laryngeal cramp in the material alteration of some organ, shows that in every case a damming up of the blood in the brain and its meninges takes place, resulting in a more or less considerable hyperæmia. He cites, as the grounds for this process: first, craniotabes, or rachitis of the upper cervical vertebræ; second, everything that is capable of exerting permanent pressure on the veins of the neck, especially at the upper opening into the thorax, such as tumors, pseudoplasms, excessive deposits of fat, hypertrophy of the thymus gland; third, rachitis of the thorax; fourth, various causes which are accompanied with sudden or prolonged passive congestion of the brain, the meninges, or the medulla oblongata, or with more or less continuous congestion of and consequent injury to the nerve structures of those parts and especially of the pneumogastric nerve.

Craniotabes has already been discussed. Rachitis of the thorax can only be construed as being of any consequence in the manner I have explained. If the irritability of the nervous system is not materially heightened, no considerable disturbance of the function of the pulmonary branches of the pneumogastric nerve and of the recurrent laryngeal branch can take place.

If passive hyperæmia of the brain, of the origin of the pneumogastric nerves or of the medulla oblongata, were sufficient to produce spasm of the glottis, the latter could hardly fail to occur in cases of insufficiency of the valves of the heart, or of serious interference with respiration resulting from diffuse inflammation or serious compression of one or both lungs, due to tumors, exudations, transudations, the escape of air into the pleural cavity, or, in case of tumors of the larynx, in croupous laryngitis, etc.

According to Loeschner, it is only while the fontanelles are open, or there are soft spots of bone, that the passive hyperæmia of the brain, etc., is capable of attaining a sufficiently high grade to produce such a degree of irritation and pressure on certain definite portions of the nerve centres as would result in laryngeal cramp. It appears to me that as long as the walls of the cranium are distensible, the degree of pressure on the brain and medulla oblongata can at least not be greater than when the roof of the cranium is closed. On the contrary, in the latter event, a less degree of hyperæmia would be capable of producing the manifestations in question. Loeschner himself admits that cases of spasm of the glottis occur after closure of the cranium, and in fact observations of this kind are not so very rare. Aside from this, we must be careful in interpreting conditions found in post-mortem examinations of this kind. If we find passive hyperæmia of the brain and its membranes, and of the medulla oblongata, transudations between the membranes, in the substance of the brain and in the ventricles, or rupture of capillaries and effusion of blood, all this does not prove that these conditions preceded the laryngeal spasm. They may just as well have been the result of the spasm and the cause of the fatal termination—at all events, the spasm must have materially aggravated these conditions if they existed. I am equally unwilling to admit that the livid hue of the face during the spasm indicates an antecedent passive hyperæmia within the cavity of the cranium. On the contrary, I have always found this cyanosis to occur secondarily when the cramp had already called forth some of the evidences of suffocation.

Material diseases of the brain and its membranes and of the medulla oblongata are certainly seldom to be regarded, in and of themselves, as the cause of spasm of the glottis. I, at least, have never observed the latter in such cases. When we remember what an irritation is exercised on the nerve ganglia in question by a basilar meningitis, and how this irritation is increased by transudation into the ventricles and œdema of the substance of the brain, we cannot but wonder that we do not see laryngeal cramp appear, either at the beginning or in the course of the process. In such cases, however, the irritation of the central portion of the pneumogastric nerve causes no spasm of the glottis, but at the height of the process induces a retardation of the pulse and superficial breathing, which, if the lack of oxygen grows too considerable, results in deep sighing inspirations. The fact, however, that this disease exerts its special influence on the laryngeal nerves is shown by the shrill cry peculiar to this disease.

Inflammatory processes in the lungs are liable to be followed by a decided abatement or disappearance of the spasm. It even appears that this is very rarely developed in lungs which are the seat of any pathological process. On the one hand, this experience argues against the theory that passive hyperæmia in the cranial cavity, which is so frequent a result of diseases of the lungs, is the cause of spasm of the glottis. On the other hand, the facts before us may easily be explained as follows. In case of anything like extensive disease of the lungs, the breathing of those parts that remain normal is materially increased to preserve the equilibrium. This heightened energy is more difficult to throw out of its rhythm than the breathing under normal circumstances or the weakened breathing of rachitis.

Catarrhs of the larynx, of the trachea, of the bronchi and their branches, are, of themselves, not capable of producing spasm of the glottis by means of reflex irritation. But when the cramp already exists, these catarrhs may awaken it, or, if it is already disappearing, may aggravate it again, especially through paroxysms of coughing.

The question whether dentition is capable of causing attacks of spasm of the glottis through reflex irritation, has never yet been settled by strict proof, which, indeed, would be difficult to carry out.

With regard to the thymus gland as a cause of this disease, we may safely join the ranks with Bednar, Friedleben, and others, who declare that it has nothing to do with this spasm. This gland has often been found swelled and diseased without spasm of the glottis, and has often failed to be affected when spasm was present, the two being sometimes also seen associated. By its anatomical position it is entirely incapable of calling forth the disease in question or of favoring its development. Care should here be taken not to diagnosticate the condition of things during life by the results of post-mortem examination. It might very easily be that the swelling of the thymus found after death had only been developed as the result of the attacks of cramp.

It is a different thing with regard to the influence of swelling of the tracheal and bronchial glands. We must admit that these are capable of inducing spasm of the glottis through irritation of the pneumogastric nerve, in a manner analogous to that which existed in the case of Ley, where the recurrent laryngeal nerve was included in a ligature with the right subclavian artery, and laryngeal cramp followed. Unfortunately in most, even of the carefully observed cases, like that of Fleischmann, rachitis of the thoracic walls existed at the same time. If these glands produce this effect by pressure, the latter must be moderate and variable. Glands that were in a state of hyperplasia, whose hyperæmia varied, would be more capable of producing these intermittent attacks of cramp than if they had already undergone caseation and become rigid. Even in the latter case, however, two circumstances may still contribute to an alteration of pressure by the glands, viz., the act of respiration and the circulation of the blood.

According to Wunderlich, in easily excitable children, prolonged violent crying, or over-exertion in walking, so as materially to interfere with respiration, are capable of calling forth light attacks of spasm of the glottis.

Romberg has seen this cramp appear after the sudden vanishing of extensive chronic eruptions of the skin, and disappear again after a relapse of the same.

A small number of cases remains in which we can in no wise find a cause for the spasm, where we are obliged to accept the theory of functional disturbance of the brain and medulla oblongata, or of simple reflex manifestations, according to Marshall Hall, and others, until such time as we may perhaps be able to demonstrate material changes as the basis of the disease. Under this category, probably, come most of the manifestations of spasm of the glottis in children over three years of age. It is also possible that, in some instances, swelling of the tracheal bronchial glands may be overlooked.

In spasm of the glottis in adults, rachitis—the most important occasion of the trouble during the age of childhood—must be left out of account. In the majority of cases we must here suppose that there is an abnormally heightened excitability of the nervous system. For this reason the disease is chiefly observed in the female sex. It may, in the first place, be caused directly from the brain, through lively emotional impressions, though, even here, the altered activity of the lungs and heart may not be left out of the account. Let us picture to ourselves the lightest grade of this process, when, in an excitable individual, as the result of anxiety or fright, the respiration becomes irregular, the action of the heart accelerated and the voice is lost; on the abatement of the cramp, then, the normal relations of the body are re-established through long, deep inspirations. At the same time material lesions of the brain and chronic meningitis have also been observed as the basis of laryngeal cramp.

Local over-irritation of the larynx, through prolonged loud speaking, may cause spasm of the glottis in persons predisposed to it. Tumors in the larynx, which are movable or capable of a variable degree of swelling, and foreign bodies, may be the cause of this disease. Every physician must have seen the artificially produced spasm often enough, in making applications to the larynx, especially as soon as the brush or sponge touches the vocal cords. In such cases the cramp is most quickly relieved by deep, powerful inspirations. Aside from this, the malady has been seen to develop itself in diseases of the epiglottis and in ulceration and abscesses of the larynx.

Reflex irritation, in any event, plays a more important rôle in the production of spasm of the glottis in adults than in children, notwithstanding that Marshall Hall lays the chief stress on this method of causation in children and endeavors to refer almost all cases to reflex manifestations. At all events, in perhaps the majority of instances, no other explanation is at present at our command. The principal basis of these reflex manifestations is found in affections of the female sexual organs, especially of the uterus. Persons thus affected may suddenly be seized with a sense of oppression, and become voiceless, even while you are talking with them, especially if they grow excited or have been moving quickly. These manifestations vanish again completely within a few seconds or minutes.

Conditions of irritation, especially foreign bodies in the pharynx or œsophagus, are capable of producing spasm of the glottis through reflex action.

Tumors and aneurisms which press upon one recurrent laryngeal nerve may, by hindering the movements of the corresponding vocal cord, produce hoarseness and a certain degree of insufficiency of breathing, which, however, is permanent and cannot be mistaken for spasm of the glottis.

Sometimes we see this cramp associated with pulmonary tuberculosis, probably accompanied by a simultaneous affection of the tracheal and bronchial glands, with epilepsy, chorea, tetanus, or hydrophobia. It has also been observed in lead poisoning.

Symptoms and Course.

These present differences, according as they are observed in children or adults. The reason for this is, that at first the glottis of the child is shorter and narrower, so that its constriction, on spasmodic action of the appropriate muscles, may be carried to a higher degree than in the adult, because the cartilaginous framework of the larynx is more yielding, and especially because the arytenoid cartilages can be pressed more firmly and uniformly against one another. Consequently the results of spasm of the glottis are most serious in the youngest children and diminish more and more with advancing years.

In children the symptoms arise as follows: the individuals attacked may be apparently well, or they may be already suffering from rachitis or scrofula, or be reduced by some other disease or unfavorable conditions of life. Bodily weakness carries with it, as a condition, the fact that all bodily activity is more readily hindered in its course than in vigorous children.

The attacks occur without warning. If in some instances catarrh of the larynx or of the bronchi has preceded it, this by no means belongs to the picture of spasm of the glottis. The lightest attacks consist in whistling or crowing inspiration. If this occurs during the night, the children go to sleep again immediately, and those belonging to them have no knowledge of what has taken place. In this way it may happen that such attacks are not noticed for weeks, unless they also arise, distinctly, during the day. The supposition that spasm of the glottis has a special predilection for the night season, is an error held by very many. Undoubtedly laryngeal catarrh, which begins with an acute swelling of the mucous membrane, and simulates croupous laryngitis, has often been mistaken for spasm of the glottis. If the attack occurs during the day, the children are seen to grow restless. When they sit up, or are held, they throw themselves backward, grow pale, and make uneasy movements with their extremities. Sometimes they roll up their eyes for a little while and turn in their thumbs for a moment. Such an attack lasts a few seconds. Immediately after it children are cross, and smaller ones cry. In a little while, however, everything is all right, and older children resume their play. Such attacks, too, are often not relieved for some time, because the attendants do not recognize them, and give the children credit for being naughty, the more so because the spasm readily occurs when children are excited.

If stronger attacks are developed, they generally also begin with whistling or crowing inspiration, following a momentary arrest of respiration, which is due, not to the condition of the larynx, but to the disturbed respiratory movements of the lungs. More rarely a few irregular, laborious, and audible expirations precede the attack. Sometimes these follow the whistling inspirations, which may occur once or several times in succession. Whether the whistling inspiration alone has taken place, or whatever may have been the order of succession of the spasmodic inspiratory and expiratory movements, there now follows a more or less complete closure of the glottis. If the closure is complete, an entire arrest of respiration takes place at once. The thorax, the diaphragm, the anterior abdominal muscles remain immovable. If the closure of the glottis is not complete, we may observe single laborious attempts at respiration on the part of the muscles concerned. At the beginning of an attack children are generally pale, and show in their faces an expression of great anxiety. Then they throw their heads back, in order to make respiration easier, the face becomes cyanotic, a cold sweat covers the forehead, the mouth is open, and the cyanotic lips, especially in little children, are put up as if they wanted to sip something. The dilatation of the nostrils helps to indicate the insufficiency of breathing. The heart's action is at first irregular and powerful; then it grows weaker and more rapid; the pulse grows small. The more severe the attack, the earlier and the more complete is the lack of consciousness. The eyes are turned up, and the hands are closed or the thumbs turned in, while the other fingers are sometimes extended and still. Not unfrequently there is a tonic cramp of the lower extremities, generally with distinct abduction of the big toe. Sometimes the wrists are bent inwards, and some have observed the feet fixed in the position of talipes varus. In still higher grades of the spasm, general tonic and clonic cramps of the body may supervene. Occasionally there is an involuntary discharge of urine and fæces during the attack. In view of the overloading of the venous system, the body, and especially the extremities, are, as a rule, cool and livid.

Such an attack lasts from a few seconds to at most two minutes. All reports of a longer duration probably depend on the fact that a succession of attacks, shortly following each other, occupied a certain period of time. As a rule, the lighter an attack is, the shorter it will be. At the same time there are short attacks which quickly reach an alarming height, and may terminate fatally the first or the second time, before help can be called. I believe that some of those cases where children are put to bed apparently well and are afterwards found dead, with evidences of cyanosis, belong in this category.

Some years ago I witnessed the following occurrence in a fat boy, six months old. The mother, a healthy woman, nursed the child herself. One evening, as she gave it the breast, it had a feeble spasm of the glottis, after which it went on nursing. Presently the child was seized with a more vigorous attack, and sank backward; whereupon I was called. I arrived there within fifteen minutes. Under the supposition that the child was asleep, he had been laid in his bed. On examination, I found him cyanotic and dead. Inspection of the body, and especially of the extremities, showed no signs of any other cramp than that of the glottis. No autopsy was permitted. In this case so complete and rapid a closure of the glottis seems to have taken place as to have left no time for the development of secondary manifestations.

The attack ends with one or more crying or whistling inspirations, which are sometimes stronger and at others very weak. The respiration, which is at first still irregular, soon becomes rhythmical, consciousness returns, if it has been lost, the heart's action becomes stronger and more regular, the cyanosis gives place at first to a pale and afterwards to the normal color. Some children sleep for a while after the attack, and, if it has not been too severe or repeated too often, wake again quite restored. If the attack has been quite severe, slight twitchings of the body may sometimes be observed during the sleep that follows. If children do not sleep after the spasm, they remain cross and peevish for a while, want to lie down or be held, cry and scream. But they soon recover, and act as if nothing had happened. If the attacks are repeated often, and especially at short intervals, the children remain weary and exhausted, want to go to bed, and show a striking degree of irritability to the slightest external influences. It may happen that a mere touch of the body is then enough to call forth a new attack. Now and then, on the other hand, a child shows great powers of resistance to these impressions, and immediately after the close of an attack seems as fresh and cheerful as before.

The disease may terminate with a single weaker or severer attack, the child become perfectly well, or be carried off in the spasm. The disease may gradually increase, the attacks become more frequent and more severe, and, after months, terminate in complete recovery. At the same time we must remember that every attack may end in death. The more quickly the paroxysms recur, the lighter are they likely to be. They may not, however, always be of the same character in the same case. A number of light paroxysms—sometimes from thirty to forty in twenty-four hours—may occur, with here and there a severe one, or the latter may be in the majority ; or the disease may consist of a larger or smaller number of only light or only severe attacks. Some children, especially when their age is no longer to be counted by months, are capable of enduring many hundred attacks, the most of which, it is true, must be of the lighter variety, without their health being permanently imperilled.

The attack gives way through the disappearance of the spasm, and, beyond all doubt, the constantly increasing lack of oxygen in the brain and medulla oblongata materially contributes to this end by inciting to powerful inspirations. If the case terminates fatally, this takes place at the height of the attack, and may result from complete and permanent closure of the glottis ; whereupon the cyanosis and carbonic acid poisoning increase, the child grows more and more exhausted, and never comes to itself again ; or death may occur suddenly, in the midst of severe general convulsions. The cause may be an acute transudation, or the effusion of blood into the brain, or general prostration of the nerve centres, if the cramp has been often repeated, or the child was in a very poor state to begin with.

The circumstances which may give rise to a recurrence of the cramp have already been discussed.

In order to insure complete disappearance of spasm of the glottis, it is important that the fundamental disease upon which it rests should be cured, this disease, in the majority of cases, being rachitis. If the children still have vigor enough to overcome the disease, and if we can place them under favorable circumstances, especially with reference to nutrition, very simple medication and baths are enough to put them in the way of recovery from rachitis, and to make the recurrences of laryngeal cramp rarer and weaker, until they finally cease. If the fundamental diseases causing the latter are such as cannot be cured, then the cramp will persist and eventually induce a fatal termination.

If the spasm has abated, so that it occurs but seldom and in light paroxysms, its intensity may again be increased if the causes are renewed or are given an opportunity again to exert a greater influence. Furthermore, every process which is calculated to lower the powers of the body—such, for instance, as profuse or protracted diarrhœa—may increase the intensity of the spasm. This experience also furnishes an argument against the theory that swelling of the thymus gland may be a cause of spasm of the glottis. Friedleben's observations prove that the leaner and more miserable the body and the more diminished its powers, the smaller in volume is the thymus gland.

As children grow older, the tendency to the development of laryngeal cramp diminishes. The principal reason for this may be, that they outgrow the period when rachitis is likely to exist. Even though the attacks may be tolerably frequent, however, they diminish in severity, because with increasing age the larynx grows wider, its cartilages firmer, and the irritability of the nervous system grows less. As soon as the attack begins, and children feel that respiration is becoming irregular, they begin to complain and want to lie down. Whistling inspiration does not occur or is insignificant. Neither is the labored expiration accompanied with any noise. The face grows pale, but little cyanotic. Patients complain of a sense of suffocation and inability to swallow. The voice is very feeble and speech laborious. General convulsions do not seem to have been observed at this age. The attacks last but a few seconds, children then getting up and being as cheerful as before. It is only when the attack has been often repeated, especially at too short intervals, that they appear exhausted and fretful. Recovery generally follows in a few weeks, though in a few rare cases a fatal termination is said to have been observed.

In adults, spasm of the glottis is most likely to occur at the period of sexual development, and is much more frequent in the female than in the male sex. With increasing years this affection is more and more rarely observed. As the larynx becomes more developed and the cartilages more firmly fixed, the attacks assume a milder form. The lightest of them are characterized by a sense of moderate constriction in the glottis. Patients state that it feels as if some one were pressing the glottis together from without. These sensations may be very transitory or last for some time. A sudden loss of voice may also occur without this feeling of constriction in the glottis. This may disappear as quickly as it appeared, or may last for days, weeks, months, and years, with slight interruptions. Wunderlich reports the case of a woman who claims to have been without a voice for twenty years, with a few interruptions. As soon as venesection was performed upon her and she saw the blood flow, the spasm ceased for days or weeks, when it would again return. Sometimes the attack begins, as in children, with whistling, crowing inspiration, noisy expiration, great restlessness, and slight twitchings. Consciousness may remain entirely undisturbed or it may be lost, even in mild cases, and then manifestations of hysterical disturbance of mind often appear. If the spasm increases and complete closure of the glottis takes place, which is certainly very rare, the symptoms do not differ from those found in children. The paroxysm may reach an equally high grade, causing the same cyanosis, the same general convulsive manifestations. It lasts from a few seconds to a few minutes, and may be repeated after longer or shorter intervals.

Another very troublesome form of spasm of the glottis, in adults, consists in cramp-like inspirations, which are repeated at short intervals, and are interrupted by violent, convulsive expirations with a shrill, piercing tone, like that of a cough. Such attacks may last a few seconds, or be continued for more than a quarter of an hour. The more severe paroxysms are always associated with more or less serious passive hyperæmia of the brain and its membranes. If these attacks are not too severe, and the entire disease does not last too long, patients are not especially affected by them. When they do last for some time, however, disturbance of the general health, loss of strength, and emaciation ensue.

Complications.

The complications of spasm of the glottis may be manifold. In severe attacks, transudations may occur between the membranes of the brain and into the ventricles, or effusions already existing may increase. If sudden death does not take place, the children die, after a longer or shorter period, with the symptoms of gradually increasing pressure on the brain. Or effusions of blood may take place between the membranes or into the brain, which, according to their situation and size, may cause the manifestations of unilateral paralysis. The duration of the latter is variable.

Ruehle has seen meningitis complicated with spasm of the glottis.

Catarrh of the larynx and of the air-passages lying below it may occur in the course of laryngeal cramp. The cough resulting therefrom may become the exciting cause for paroxysms of spasm which already exists. In rare instances, croupous inflammation of the larynx may become associated with spasm of the glottis, in which case the disease may terminate fatally.

Inflammation of the lungs sometimes appears as a complication. The laryngeal spasm is then likely to grow weaker or to disappear entirely. This disease has, furthermore, been found associated with chronic pulmonary affections, acute œdema of the lungs, heart disease of various kinds, hyperplasia of the spleen, and intestinal catarrh.

Pathological Anatomy.

The results arrived at in this department, so far as the laryngeal cramp itself is concerned, have thus far remained entirely negative. No alterations have been discovered in the nerves nor in the structure of the muscles or mucous membrane. The latter has, in some cases, been found moderately reddened.

Amongst the conditions which have always been found present are a greater or less degree of hyperæmia of the brain membranes and of the brain itself, sometimes effusion of blood, œdema of the brain, transudation between the membranes or into the ventricles. In rare instances inflammation, hypertrophy, or partial softening of the brain has been seen. Softening of the medulla oblongata has also, very occasionally, been demonstrated.

Sometimes it has been possible, after death, to demonstrate the traces of catarrh of the respiratory organs. Occasionally a croupous exudation on the mucous membrane of the larynx or the trachea, ulcerations and abscesses in the larynx have been found.

Swelling of the thymus gland, with serious reddening of the same, belongs amongst the occurrences that are not exactly frequent. Sometimes its color is quite pale. Chronic inflammation of this organ, with caseation, is very rarely encountered.

Loeschner, in one case, observed an abscess, the size of a bean, over the hyoid bone.

A condition of not so great rarity is more or less serious swelling of the tracheal and bronchial glands, with partial or complete caseation.

Hypertrophy of the left ventricle, or dilatation of the right ventricle of the heart, or patency of the fœtal circulatory passages are all sometimes found. Pericarditis is extremely rare. Loeschner claims to have found the thoracic organs uniformly somewhat anæmic in his post-mortems. Barthez and Rilliet and Bednar assert, on the contrary, that in the autopsies made by them the heart was never quite empty, but, as a rule, was overfilled with black, fluid blood, as were also the larger vessels of the thoracic cavity.

The lungs are usually emphysematous to a variable degree and extent, as the result of the spasmodic respiration. Not unfrequently œdema is likewise present. Sometimes they give evidence of the various forms of acute or chronic inflammation, with or without chronic tuberculosis and excavations.

Redness and swelling of the gastric and intestinal mucous membrane, hyperplasia and caseation of the mesenteric and retroperitoneal glands are met with now and then, the latter being generally associated with the same process in the tracheal and bronchial glands.

Hyperplasia of the spleen has seldom been demonstrated.

Swelling, with a yellowish or yellowish gray color of the liver and a considerable deposit of fat within its cells, is a frequent condition in spasm of the glottis. In some few instances diffuse chronic tuberculosis has been demonstrated.

As a matter of course, the products of rachitis are the ones most frequently found in the dead subject, and can chiefly be demonstrated in the bones. In many cases craniotabes may also be found, though this is by no means the rule.

Diagnosis.

The symptoms of spasm of the glottis are so characteristic that one can hardly believe any other disease capable of being confounded with it. The sudden beginning of the attack, the whistling or crowing inspiration, the noisy expiration which sometimes occurs, the increasing cyanosis, the frequent loss of consciousness, the partial or general convulsive manifestations at the height of the attack, the feverless course and short duration of the disease, the absence of cough, the termination of the attack through convulsive, crowing inspiration, and the free intermissions, constitute a perfectly clear picture of this disease.

In spite of this, it has, not unfrequently, been confounded with croupous laryngitis. It has also been asserted that there were cases where one might waver in his diagnosis between spasm and œdema of the glottis or whooping-cough.

The first step towards a correct diagnosis is a careful examination of the general constitution of the body and the condition of its individual organs. If, then, a hindrance of respiration, a certain degree of respiratory insufficiency, is found to depend on some material disease of the respiratory organs, the heart, the brain and its membranes, or the medulla oblongata, no one will be able to assert that he is dealing with a case of spasm of the glottis unless it is clearly characterized by the above-named symptoms. At the same time, spasm of the glottis may be associated with diseases of this kind. If there is no disease of other organs, and the symptoms appear as described, then one is dealing with a case of simple laryngeal cramp. The existence of the latter is rendered the more probable if rachitis is found to be present. Any considerable swelling of the tracheal glands may be determined with confidence by the dulness which it causes in the angle between the clavicle and sternum.

Croupous laryngitis may be distinguished from spasm by the following characteristics: it never arises suddenly, but, if carefully watched, will always be found to be preceded by a period of variable length, during which the child is ill at ease and suffers with catarrh of the respiratory organs. The disease begins with a dry, barking cough, which, as it increases in severity, alternates with spasmodic, whistling inspiration, but which neither begins nor ends with this symptom.

The attacks last longer than those of spasm. There are no convulsive manifestations whatever. The repetition of the attacks may result in great difficulty of breathing, but is not followed by direct suffocation. As the attacks become more frequent, the passive hyperæmia of the venous system, especially in the cavity of the cranium, is continuously augmented; and the same holds true with regard to carbonic acid poisoning. If the latter reaches a certain grade, consciousness, which up to that time has remained clear during the attacks, may be partially or wholly lost. At the same time a loss of sensation sets in. As cyanosis increases, the spasmodic inspiration becomes more violent. The disease is generally associated with fever, and shows no well-marked intermissions. A certain degree of respiratory insufficiency persists between the lighter attacks. In the intervals between the severer attacks the difficult breathing may be heard even in the next room, characterized by laborious and noisy inspiration and expiration. Auscultation of the larynx distinctly indicates the hindrance to the inspiratory current of air, while after spasm of the glottis the normal relations are found to exist. In older children all doubt may be removed, from the first, by laryngoscopic examination; while the existence of an affection in the pharynx is no evidence, either pro or con.

Croupous laryngitis is, therefore, not easily to be confounded with spasm of the glottis. But a much freer interchange has been implied by writers between the latter and the catarrhal form of laryngeal inflammation, which has had the names of laryngismus stridulus, pseudo-croup, etc., imposed upon it. This disease may arise suddenly, or after catarrhal affections of the bronchi and their branches, cold in the head, etc., have shown themselves for several days. I have often observed that in children, for years at a time, every lively catarrh of the nasal mucous membrane spread downwards to the larynx and here produced catarrhal inflammation. The symptoms of the latter resemble, in general, those of the croupous process, except that here the respiratory insufficiency by no means reaches as high a grade, provided that acute œdema does not supervene. The attack may pass off in a few minutes or last half an hour. It may be repeated several times, and so prolong the entire course to a number of hours or even days. In the majority of cases the attack comes on during the night, after a child has been put to bed apparently well, and often shows a disposition to return on the following night. The intervals are never perfectly well defined, but the noisy respiration always shows a certain degree of insufficiency, though the children can still sleep quietly. After the disease has run its course, children are weary and used up, and do not recuperate for several days. There hardly seems to be any fever, nor can any considerable degree of passive hyperæmia and cyanosis be demonstrated. On the other hand, the dry barking cough, interrupted by spasmodic, whistling respiration, is characteristic.

Œdema of the glottis is very rare in children. As a primary affection, it only arises when, in swallowing, hot or caustic substances pass into the larynx. Secondarily, it may associate itself with croupous, diphtheritic, and severe acute catarrhal inflammation of the laryngeal mucous membrane. It is generally developed gradually, though sometimes quite acutely. If one is experienced, he can, with his finger, feel the swollen and rigid mucous membrane of the epiglottis and arytenoid cartilages through the mouth. Generally the mucous membrane of the fauces and velum is also somewhat swollen and pale. Swallowing is difficult. The œdema itself causes no fever nor cough. On the other hand, there is a tormenting sensation of insufficiency of breath, the respiration is laborious, inspiration and expiration are both noisy, and inspiration, particularly, is long-drawn and somewhat whistling. No free intervals exist. Auscultation of the larynx distinctly indicates a hindrance to respiration as long as the disease lasts.

Whooping-cough never arises suddenly, but always has a catarrhal initial stage of some weeks in length. The paroxysm of coughing itself is not sudden, but is often felt by the child ten minutes in advance. It is characterized by a succession of severe coughs following one another rapidly, which, if of a high grade, are interrupted by long-drawn, whistling inspirations. The paroxysm lasts from a few seconds to several minutes, the face becoming red and turgid. The younger the children are, the more likely are they to become unconscious during the attack, and to remain so even for a few minutes afterwards. Children, during the first years of life, may suddenly perish at the height of a severe paroxysm, through transudation into the brain and the ventricles, or through cerebral apoplexy. No such lasting closure of the glottis takes place as might produce suffocation. No fever is connected with the attacks. The intervals are perfectly free if the disease is not complicated with other processes.

In adults, spasm of the glottis can only be confounded with material diseases of the larynx, such as ulcerations, abscesses, diseases of the epiglottis, œdema of the glottis, polypi or foreign bodies in the larynx. More careful manual examination, the use of the laryngeal mirror, and auscultation of the larynx must protect us against all error.

Prognosis.

This is variously given by different writers, being also often based upon a rather indefinite diagnosis. Aside from this, however, it must vary according to the age, especially as between children and adults, and according to the intensity of the attacks.

First, as regards the age of childhood, Ruehle considers the prognosis, in individual cases, to be doubtful, and on the whole to be unfavorable. Friedreich designates the severe forms only as being doubtful. According to Wunderlich, about one-third of all those attacked, and the majority of those visited with severe attacks, die.

Barthez and Rilliet	had	8	deaths among	9	patients.
Hérard	"	6	"	7	"
Reid	"	115	"	289	"
Lorent, in boys,	"	45	"	100	"
" in girls,	"	32	"	100	"
Salathé	"	2	"	24	"

Bouchut declares that a little more than half die, and Steiner is of the opinion that the great majority die.

According to my experience, if we include the lightest cases, which we certainly should, the prognosis is, on the whole, favorable.

The best prospect is afforded, in general, by those cases, which do not concern too young children, when the patients are, on the whole, vigorous, when the attacks are weak and run their course briefly and without general convulsions, especially if they consist of one or but a few whistling inspirations. Furthermore, if the degree of cyanosis is slight, consciousness is retained, and the intervals between the paroxysms are quite long. Some writers assert that paroxysms which last over two minutes are fatal.

In individual cases the prognosis must be based upon the diseases which constitute the foundation for the glottic spasm. The most favorable cases are those in which it is developed as the result of rachitis. If the strength of the patient is to any degree sufficient, if one is able to change the conditions of life, especially the nourishment, satisfactorily, if the directions given are strictly followed, one will see favorable results in the majority of cases. It is true that weeks and months often pass before one can observe any material change for the better. But there are also cases where a change is effected pretty soon, especially if the alteration in the circumstances of life, aided by therapeutics, has been very radical.

A striking example of this may be reported in brief. In the spring of 1873 I was called to a country-seat. It was said that the son of the proprietor, a boy ten months old, was suffering from hydrocephalus and convulsive attacks, and was becoming idiotic. The father of the proprietor, his mother, and one sister had suffered from mental disease, the former having died in this condition. The parents of this boy, who was their first child, were slender in build but otherwise healthy.

Examination showed that the child was suffering from rachitis, which was in every respect highly developed, and from spasm of the glottis, which was not rarely associated with general convulsive seizures. The laryngeal cramps were capable of being called forth by the most insignificant causes, were only of short duration, but very numerous. The child had had a wet-nurse up to its fourth month, and since then had been fed chiefly on articles containing flour. No teeth were as yet developed. In addition to giving cod-liver oil, iodide of iron, baths with salt and malt, I rigidly prescribed the diet. The child was only permitted at certain regular intervals, to be fed on the best of milk, beef broth, and the yolks of eggs. In fourteen days the father wrote me that the attacks were much less frequent and weaker. Six weeks later he brought me the information, personally, that the spasm had entirely disappeared. When the child was a year old he had cut several teeth without any trouble. Since that time, in addition to his other diet, I have allowed the parents to give him good meat. The child walks now, is strong, and intellectually as well developed as the best children of his age.

As soon, in such cases, as the appearance of rickets gives way, the spasm of the glottis also moderates, and, with the progress toward recovery, naturally disappears much earlier than the former.

Scrofula, which, in the cases belonging here, finds its expression in swelling and caseation of the tracheal glands, renders the prognosis more doubtful. At the same time this process, too, may recede, though in the minority of cases, and then the spasm of the glottis dependent on it will disappear.

Material diseases of the brain, its membranes, and the medulla oblongata, affect the prognosis unfavorably, and, if they are incurable, fatally.

Those cases which are brought about by means of reflex action admit of a favorable prognosis if the cause can be removed, not otherwise.

Processes which are capable of lowering the powers of the patient, during the progress of spasm of the glottis, vitiate the prognosis. If the spasm has already moderated, or is in the act of disappearing, any weakening of the powers may increase it again or call it forth anew.

All complications of laryngeal cramp, which have been spoken of above, and which, especially by other writers, have been adduced as causes thereof, make the prognosis more unfavorable on that account, and, if they are incurable, generally make it fatal. An exception to this is found in acute inflammation of the lungs, which usually effects a material abatement and even complete disappearance of the laryngeal cramp.

On the whole, the affection is said to admit of a more favorable prognosis in the female than in the male.

In adults, the prognosis depends upon the pathological condition of the organs which cause the spasm. The more obstinate the attacks are, and the more they remain confined to the larynx, the more doubtful is the prognosis. If the material disturbances lying at the foundation are incurable, then the prognosis for the spasm is bad, and may, under some circumstances, be fatal; if they are removable, the prognosis is favorable. It is well, however, to be cautious in one's predictions. Cases have terminated fatally when the physicians did not in the least expect it. Among adults, too, the female sex has the advantage of a more favorable prognosis, because the sexual system plays an important rôle ; whereas in men it is usually a question of some severe organic disease.

Treatment.

The treatment likewise presents different features according as the patients are children or adults. Aside from this, it is twofold in both classes, being directed both against the conditions lying at the foundation of the spasm and against the attacks themselves. Accordingly, in the majority of cases occurring in children we have to combat rachitis, and, in the next largest number, scrofula. Little children should be kept on a good and adequate supply of breast milk. If this is impossible, then ass's milk or cow's milk should be given, diluted to a degree proportionate to the age of the child. If the child is over six months of age, meat broth should be given, besides the milk. As soon as the child has cut some incisor teeth in both jaws it should have good, underdone meat, finely minced. If the patient is very poorly nourished, some yellow of egg may be given earlier. Aside from this, the administration of some Hungarian wine, or good French red wine, is to be recommended. Until the period of life in which children are subject to rachitis is passed, they should receive no nourishment containing flour, and afterwards rye-bread only in very small quantities. Until the eighth year their main nourishment should consist of milk and meat.

Children should not be kept too warm in bed ; after they are six months old they should be laid on a firm pillow [or mattrass], not filled with feathers, and should be washed with cold water. If not able to sit up alone, they should not be propped up in chairs nor carried in a sitting posture, especially not if they are rachitic, because in this way slight deviations from the normal axis of the spinal column may be favored, and new occasion thus be given for the development of spasm of the glottis through hindrance of respiration. Lying down is of no sort of disadvantage to children with craniotabes. All the functions of the body are, as far as possible, to be kept in order, care being taken to prevent constipation or diarrhœa, by the appropriate means. It is important that there should be plenty of fresh air in the room, and that, when the weather is good, children should be out-doors as much as possible. All external causes which might excite the nervous system should be avoided and children should be given abundant time for sleep.

The best remedies we can administer against rachitis and scrofula are cod-liver oil, syrup of the iodide of iron, and baths containing salt and malt. I have never seen any result from the administration of preparations of lime in rachitis.

If distinct organic diseases constitute the cause of spasm of the glottis, or complicate it, the treatment necessary to insure their cure must be adopted. If these diseases are incurable, we must endeavor to lessen their influence on the production of the spasm.

Inasmuch as some authors regard difficult dentition as one of the causes of spasm of the glottis, West, Bouchut, Barthez and Rilliet, and some others, have advised lancing the gums under these circumstances. Many others, as well as myself, have never seen any result from this operation, either in this or any other pathological condition ; consequently I can in no wise recommend it.

Light cases receive no treatment, partly because it is not needed, but still more because, owing to their brevity, it is impossible.

If the attack lasts longer, so that there is time for interference, the first thing to be done is to use external counter-irritants. Sinapisms may be applied, various portions of the body may be rubbed with spirits of mustard, the face may be sprinkled with cold water, or the surface of the body may be rubbed with it. If they are at hand, strong liquor ammoniæ or ether may be held under the nose. Simpson proposed the inhalation of chloroform, which, according to my opinion and that of others, should be employed with great caution, notwithstanding that Cox, Smage, and West claim always to have secured prompt results, and never to have seen any ill effects therefrom. Perhaps hypodermic injections of morphine might be of service. Mayer, in the "Petersburger medicinische Zeitschrift," recommends subcutaneous injections of atropine.

If the cyanosis assumes a higher grade, consciousness is lost, and general convulsive attacks set in, cold affusions over the head and neck must be used—the best way of giving them being with the patient placed in a full warm bath of from 90° to 95° Fahr. If there is not enough warm water at hand for this, he should at least have a warm foot-bath at the same time. If the patient is in a hospital the powerful application of an induction-current to the phrenic nerves may be of benefit. Some have advised the administration of an emetic, which could hardly be taken during a severe attack. If it is desired to try this, which is somewhat hazardous, a hypodermic injection of a solution of apomorphine should be used, which, if made strong enough, is likely to act promptly and effectively. If the mouth is open, the attempt may be made to excite vomiting by tickling the pharynx and root of the tongue.

If the cyanosis remains of a high grade, unconsciousness continues, and the spasm of the glottis remains of the same intensity, one may be forced, in very vigorous, full-blooded children, to moderate the passive hyperæmia of the brain and its membranes by the application of leeches. This necessity, however, will only arise in the rarest cases, and the abstraction of blood always remains a doubtful measure, as well with reference to the attack as to the further course of the disease.

Marshall Hall, Friedreich, Wunderlich, and others recommend tracheotomy, in the extremest emergency, when suffocation is threatening. Aside from the fact that, unless the patient happens to be in a hospital, this operation cannot always be performed quickly enough, I have never yet learned of any favorable result that has followed it in spasm of the glottis.

As respiration is always less free, more shallow and rapid, when the patient is lying down than when the back of the thorax experiences no pressure, the paroxysms can be made easier by raising the patient up at once.

The use of certain medicaments during the attack has been recommended, provided the spasm is not so extreme that patients cannot swallow or that consciousness is lost. But as the great majority of laryngeal cramps are light, it may well be practicable to give internal remedies. The principal ones of value are musk and castor in sufficient doses. No rapid results can be expected from any other agents. Perhaps an enema of asafœtida or tobacco may be useful.

During the intervals all possible remedies have been brought into the field against this disease. The preparations of zinc, especially the cyanide, with or without calomel, have been lauded by a good many, especially by Pagenstecher. Other have recommended the preparations of copper, nitrate of silver (Heer), opium, morphine, digitalis, cherry-laurel water, hydrocyanic acid (Caspari), asafœtida, internally or in enema (Romberg), extract of belladonna (Stark), extract of artemisia (Pfeufer), extract of lactucarium, bromide of potassium (Fingerhuth), tincture of cannabis indica (Bouchut), chloride of gold (Wendt), electro-magnetic treatment (M. Meyer), enemas of tobacco, etc.

Most of these remedies may unquestionably be passed over in silence. We may perhaps except asafœtida, large doses of bromide of potassium, which must be used long and continuously, and nicotiana, which remedies are capable of decidedly moderating the irritability of the nervous system.

The most effective remedy is undoubtedly musk, which was proposed and used by Reid, and has been warmly recommended by many others, especially by Salathé. I rank castor next to this for very full-blooded children. I have obtained very good results from both drugs, particularly very material weakening of the attacks.

Of late chloral hydrate has been recommended by various parties. Rehn narrates a case in which he could demonstrate an abatement of the spasm in frequency and severity after the use of chloral for three days. Twelve days after beginning its use no more characteristic paroxysms occurred. During the succeeding days and weeks occasional, quite light manifestations appeared now and then. Others have seen no results from the use of this remedy

I consider applications of croton-oil in the neighborhood of the larynx, the use of blisters, either alone or with the simultaneous use of morphine, iodide of potassium, externally, or tincture of iodine, all to be superfluous. The results of employing the gray oxide of mercury ointment are injurious.

It is a matter of importance to improve the strength; we must not fail, in addition to appropriate nourishing diet, to administer iron, if the digestion will bear it, and especially quinine. These remedies may be continued with advantage for some time after the removal of the disease.

If patients live in a city, great advantage will be obtained from removal into pure, healthy country air. A material abatement, and even the sudden disappearance of the spasm, has often been observed as the result of this change. Aside from this, all the exciting causes which were spoken of above as capable of calling forth the cramp anew must be avoided. Among these are taking cold, errors in diet, emotional disturbances, sudden movements of the body. Children should not be awakened from sleep, and should not be allowed to take any nourishment soon after the attack, because a new paroxysm may easily be called forth, partly through the act of swallowing, and partly because, under the circumstances, some of the matters swallowed may pass into the larynx.

Daily luke-warm baths have a beneficial effect.

In adults—aside from sudden attacks of spasm of the glottis, the result of foreign bodies in the larynx or œsophagus, which must be extracted as soon as possible, and polypi, which should be removed without delay—therapeutic interference is to be adapted to the physical constitution of the patient. In full-blooded individuals we must insure copious stools and provide mild, unirritating diet. If laryngeal cramp occurs after the sudden failure of the menses, a local abstraction of blood must be undertaken. In weakly persons the strength must be built up by good diet, iron, quinine, and malt or steel baths.

Above all, the causes of the cramp must be accurately determined, and the appropriate treatment to combat them must be adopted. In moderate attacks no interference need be undertaken. In severer ones the same means are applied, in the same way as in children. Here, too, tracheotomy is recommended, and indeed its early employment is advised. Wunderlich and others claim to have seen good results from the abstraction of blood in the region of the larynx. During the intervals the entire host of anti-spasmodic and tranquillizing agents which are prescribed during the age of childhood are also recommended, as well as the proper regulation of all the conditions of a person's living. Among adults, too, our main reliance will have to be on asafœtida, bromide of potassium, musk, and castor. Some advise external local irritants in the region of the larynx, for instance, blisters, croton-oil, veratrine ointment, or even moxas and setons.

If the causes of the spasm are incurable, we are restricted to rendering the attacks as mild as may be, and to making the general condition of the patient as endurable as possible.

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